



# MEDICAL DISEASES OF WAR

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To

MY WIFE,

to whom *Medical Diseases of the War* was dedicated in 1916 as acknowledgment of all she had done for the rehabilitation of my patients at Netley and Seale Hayne Hospital, and to whose help and encouragement in the preparation of *Medical Diseases of War* I owe so much

## PREFACE TO FIRST EDITION

The first edition of my book on *Medical Diseases of the War* was published in November 1916. At that time I had had opportunities of studying the various diseases occurring on active service first as physician and neurologist to a number of military hospitals in London and to the New Zealand Hospital at Walton-on-Thames, then as a member of the Medical Advisory Committee for the Prevention of Epidemic Disease in the Mediterranean Expeditionary Force in Lemnos, and subsequently as Consulting Physician to the British Forces in Salonica. The second edition was published in March 1918 when I had had further experience as Neurologist to the third Southern General Hospital at Oxford and to the Royal Victoria Hospital at Netley. During the next fifteen months I was in command of the Seale Hayne Hospital for Functional Nervous Disorders. At the end of 1918 I had collected material from my own experience and from a study of the available literature in English, French, Italian and German for a third edition, but the Armistice made its publication unnecessary.

I have called this new edition of the book *Medical Diseases of War* instead of *Medical Diseases of the War* as I hope it may be of use to medical officers called upon to treat soldiers and civilians during the present war. I have tried to bring each chapter up to date in the light of knowledge acquired since 1919. I have omitted certain chapters from the earlier editions dealing with conditions which are unlikely to occur in the absence of an Eastern campaign. I have invited Dr H. W. Barber to write a chapter on Skin Diseases in War as no less than 30 per cent. of medical casualties in the British Expeditionary Force in the last war were the result of scabies and pediculosis.

I have also been fortunate in obtaining the help of Dr T. A. Ross in the section on psychoneuroses. The war gave us a great opportunity of studying the varying manifestations of hysteria in soldiers. By the end of 1918 we had been able to demonstrate the hysterical nature of several conditions which had hitherto been regarded as organic and we had gradually evolved methods of treatment which made it possible to do in an hour what had at first required weeks or months. This experience seems worth recording in detail again

as many of the hysterical symptoms which were common in the war are very rarely seen in peace-time, and moreover most modern psychotherapists are accustomed to slow methods of treatment which would be useless in war-time. I hope, however, that Dr. Ross's chapter on the Anxiety Neuroses will help to explain the war neuroses in the light of modern psychotherapy.

Lastly I am greatly indebted to Dr. F. A. Knott for writing the sections on the bacteriological and serological diagnosis of enteric fever and dysentery, and to Dr. A. A. Osman for his valuable criticisms on the chapter on War Nephritis.

## PREFACE TO FOURTH EDITION

In the second edition of *Medical Diseases of War* new chapters were written by Maj-Gen A. W. Stott on meningococcal fever by Col. H. B. F. Dixon on malaria, and by myself on digestive disorders. In the third edition new chapters were added on typhus fever by Dr Melville D. Mackenzie and on diphtheria by Dr E. H. R. Harnes.

In the present edition the fourth of the present war the seventh of the book originally published in 1916 the chapter on infective jaundice and infective hepatitis and the sections on sciatica and the treatment of bacillary dysentery have been rewritten, the first with the help of Col. Dixon. The section on the seborrhoeic state has been replaced by one on Dermatophytosis. Considerable alterations have been made in the sections on digestive disorders malaria, the incidence and prophylaxis of tetanus and the hysterical symptoms following concussion. Minor corrections and additions have been made to most of the remaining chapters in the light of further personal experience and the available literature up to July 1943

I am grateful to the Wellcome Museum of Medical Science, Col. C. F. Craig of New Orleans, Dr B. B. Vincent Lyon of Philadelphia, Dr F. A. Knott and Dr J. McMichael for illustrations added to the second, third and present editions. Col. Craig's micro-photographs are reproduced by kind permission of Dr S. A. Portus and Messrs. Lea and Febiger from Portus's *Diseases of the Digestive System*.

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# CONTENTS

CHAP	PAGE
I PREDISPOSING CAUSES OF WAR NEUROSES	1
II HYSTERICAL SYMPTOMS IN SOLDIERS	5
III HYSTERICAL PARALYSIS	14
IV HYSTERICAL CONTRACTURES	21
V RHEUMATISM, SCIATICA, AND HYSTERICAL POSTURES AND GAITS	45
VI HYSTERICAL TREMOR	61
VII HYSTERICAL FITS	64
VIII DISORDERS OF SPEECH	68
IX FUNCTIONAL DISORDERS OF HEARING	84
X FUNCTIONAL DISORDERS OF VISION	90
XI HYSTERICAL STUPOR AND AMNESIA	113
XII CEREBRAL AND SPINAL CONCUSSION	121
XIII EXHAUSTION RESULTING IN NEURASTHENIA	130
XIV HYPERADRENALISM AND HYPERTHYROIDISM	143
XV ANXIETY NEUROSES OF WAR ( <i>By T A Ross</i> )	149
XVI DIGESTIVE DISORDERS IN SOLDIERS	175
XVII EFFORT SYNDROME	190
XVIII TRENCH FEVER	216
XIX LOUSE BORNE TYPHUS FEVER ( <i>By Melville D Mackenzie</i> )	235
XX TYPHOID AND PARATYPHOID FEVERS	261
XXI DYSENTERY	284
XXII EPIDEMIC JAUNDICE; INFECTIVE HEPATITIS	323
XXIII MALARIA ( <i>By Col. H B F Dixon</i> )	343
XXIV MEXICO-COCCAL FEVER ( <i>By Maj Gen. A W Stoll</i> )	376
XXV DIPHTHERIA ( <i>By E H R Harris</i> )	392
XXVI TETANUS	413
XXVII WAR NEPHRITIS	437
XXVIII SKIN DISEASE IN WAR ( <i>By H W Barber</i> )	450
XXIX GAS POISONING	492
INDEX	509

# LIST OF PLATES

		<i>Facing Page</i>
<i>PLATE</i>	<i>I</i> RADIOGRAPH OF HANDS SHOWING INCREASED TRANSPARENCY OF BONES AFTER HYSTERICAL CONTRACTURE	302
<i>PLATE</i>	<i>II</i> <i>ENTAMOEBA HISTOLYTICA</i> , <i>E. COLI</i> , <i>ENDOLIMAX NANA</i> AND <i>LAMBLIA INTESTINALIS</i> .	303
<i>PLATE</i>	<i>III</i> INFECTIVE HEPATITIS	324
<i>PLATE</i>	<i>IV</i> BLOOD FILMS IN MALARIA	325
<i>PLATE</i>	<i>V</i> TEMPERATURE CHART IN BENIGN TERTIAN MALARIA WITH DEVELOPMENT PHASES OF THE PARASITE	350
<i>PLATE</i>	<i>VI</i> TEMPERATURE CHART IN QUARTAN MALARIA WITH DEVELOPMENT PHASES OF THE PARASITE	351
<i>PLATE</i>	<i>VII</i> SCABIES PEDICULOSIS CORPORIS	466
<i>PLATE</i>	<i>VIII</i> PEDICULOSIS CORPORIS VITILIGO AND PEDICULOSIS CORPORIS	467

# MEDICAL DISEASES OF WAR

## CHAPTER I

### PREDISPOSING CAUSES OF WAR NEUROSES

War neuroses accounted for about 6 per cent. of the 341,025 discharges from the British Army up to the end of April 1918. In the present war they have been no less common. The importance of the subject from a national point of view can scarcely be exaggerated.

The chief predisposing causes of war neuroses are a neuropathic or psychopathic inheritance and a previous nervous or mental breakdown. Some men are constitutionally brave others are constitutionally timid, the average man is neither. There are a few who do not know what fear is their bravery is so natural to them that it is hardly a virtue. Hunger and thirst do not depress them, wrote the Student in Arms. "Rain could not damp them. Cold could not chill them. Every hardship became a joke. They did not endure hardship they derided it. As for death, it was, in a way the greatest joke of all. But most men feel frightened when first exposed to the danger of a bombardment they perspire profusely their hands shake their hearts beat rapidly and each time they hear a shell whistling by they duck their heads. Their bravery is a genuine virtue, as it consists in the complete suppression of their natural fear. As the Student in Arms wrote, "Your teeth may chatter and your knees quake, but as long as the real you disapproves and derides this absurdity of the flesh, the composite you can carry on." Turanne was seized with trembling on the field of battle. Ah, carcass! he exclaimed, you tremble. But if you knew where I am going to take you you would tremble much more." In the course of time such men generally become more or less accustomed to the sights and sounds which at first frightened them, and the necessity of suppressing the emotion of fear becomes more and more rare. The constitutionally timid, on the other hand, are more liable than the average soldier to all war

neuroses, and if blown up by a high explosive shell, they are almost certain to develop hysterical symptoms in addition to the symptoms due to concussion. They have not the strength of will required to suppress the fear they feel, often even before they reach the danger zone, and they break down as soon as they hear the sound of distant guns. These men, well described by Buzzard as "martial misfits," are frequently able to live a normal life as civilians, but they are totally incapable of adapting themselves to a soldier's life. An officer, typical of many similar cases, obtained his commission in July 1915, he had always been nervous and subject to palpitation and diarrhoea when excited. Directly he reached the front he became sick with terror, sweated profusely and had constant palpitation and diarrhoea. At the end of a month he was quite unable to carry on.

Some martial misfits are easy to recognise by their appearance and their conversation, and the majority themselves recognise their disability. The artistic temperament is common among them, although it is of course far from being incompatible with bravery. In other cases the discovery that they are martial misfits comes as a surprise to themselves and their friends. A keen territorial officer of many years' standing, who had been fretting during the first eighteen months of the war because he had been kept at home for training purposes, discovered to his horror that he was totally incapable of facing the strain of active service at the front. He became so emotional and tremulous as soon as he approached the trenches that he had to be sent home.

Contrary to what might have been expected, soldiers from the Dominions are no less liable to functional nervous disorders than English and Scottish troops. The French probably suffered rather more than the British. Neuroses were very common in Germany, but L. R. Muller, who was consulting physician to the Turkish armies in the last war, stated that he neither saw nor heard of any cases of hysteria or other functional nervous disorders among them. Among 10,000 Serbian prisoners, who fell into German hands, exhaustion, hunger, loss of sleep and all manner of infections led to great physical weakness and emaciation, cardiac failure and oedema, and many deaths from tuberculosis, but only five cases of psychoneuroses were observed.

Men with a family history of nervous or mental disease and men who had previously suffered from some nervous or mental break-

down whatever may have been the cause were particularly liable to develop all forms of war neuroses. Out of 100 patients suffering from war neuroses Wolfsohn (1918) found that 74 had a neuropathic or psychopathic family history compared with 38 out of 100 wounded soldiers. Among the former there was a family history of insanity in 34, epilepsy in 30 and alcoholism in 50 but among the latter there was no history of insanity or epilepsy, and of alcoholism in only 24. In the same series 72 had a personal history of neuroses compared with 10 of the wounded, all of whom also suffered from mild neurasthenic symptoms. Concussion caused by an injury to the head even years before makes a man specially liable to develop functional nervous disorders, unless it was treated by more prolonged rest than is usually given. Thus 38 of Wolfsohn's 100 cases of war neuroses had previously been injured in the head, but only 12 among his 100 controls. Out of 100 neuro-psychiatric cases admitted into a Royal Naval Hospital in the present war 45 gave a family history and 32 a personal history of nervous disorders compared with 6 and 4 respectively among 50 control surgical cases. Alcoholism was rare and sexual conflicts and sexual deprivation played an insignificant rôle (Curran and Mallinson 1940).

A man with a good family history, who has never suffered from any nervous disability develops war neuroses only under very exceptional circumstances. A single explosion for example, was unlikely to give rise to symptoms apart from those due to concussion, unless he was already weakened by a long period of physical fatigue combined with severe mental strain. More frequently serious symptoms developed only after exposure to a third or fourth explosion.

Chronic alcoholism is rare in men of military age. The few alcoholics who become soldiers benefit greatly from the discipline and comparatively healthy life they lead during training. But even if they take little or no alcohol after enlisting they remain particularly liable to nervous disorders when exposed to the stress and strain of active service. It is, however, remarkable that 48 out of Wolfsohn's 100 cases of war neuroses occurred among teetotallers, and only 6 had indulged in alcoholic excess, whereas only 20 of 100 wounded were teetotallers and 16 had indulged in excess. The rum ration is abundantly justified by its stimulating effect when an attack has to be made at dawn, but many men suffering

from the early stages of various war neuroses precipitate their final breakdown by attempting to keep themselves going by means of alcohol

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## CHAPTER II

### HYSTERICAL SYMPTOMS IN SOLDIERS

Hysterical symptoms in soldiers during the war of 1914-18 could be divided into four main groups (1) those which were emotional in origin, (2) those which followed gassing (3) those which followed trivial wounds to limbs, and (4) those which followed injury to the central nervous system. As they differed greatly in the type of man involved and in prognosis they require separate consideration, especially in view of the fact that whereas the first class was, after an early period of doubt, widely recognised as being of psychological origin, the remaining classes were often regarded as organic in nature even at the end of the war. Consequently most special hospitals for functional nervous disorders had many of the first group of cases in addition to soldiers with anxiety neuroses, but comparatively few of the second and fourth groups and often none at all of the third group. On the other hand, as a result of visiting large numbers of military and V.A.D. hospitals in a consultant capacity, I was able to collect some hundreds of patients belonging to these classes, especially the third and have them transferred to Seale Hayne Hospital, where their long period of incapacity was brought to an abrupt conclusion by suitable psychotherapy.

The four different groups of hysterical symptoms occurred in quite different types of men. The first occurred almost invariably in men who were constitutionally nervous, the martial misfits. The hysterical manifestation was always associated with anxiety symptoms. The third group of symptoms occurred among quite ordinary soldiers. They never gave a personal or family history of neuroses, they had no associated anxiety symptoms, and when cured they were always able to return to active duty, unless they had been demoralised by too long residence in hospital. The second and fourth groups were intermediate, the majority of the second and a minority of the fourth occurring in men of a constitutionally neurotic type.

The development and general character of each class is discussed in this chapter. A detailed description of the symptoms will be given in subsequent chapters.

(1) **Cases with emotional origin.**—Extreme terror gives rise to certain familiar physical results, the individual becoming shaky, “paralysed with fear,” and unable to speak—“his tongue cleaves to the roof of his mouth” Under ordinary conditions the cause of fear is momentary and the physical results disappear in a few seconds But during a heavy bombardment a man often remained terrified for hours If the tremor, inability to move the legs and speechlessness persist all the time, it is natural that these symptoms of fear, which are not in any way hysterical, should so greatly impress the soldier’s mind that the idea of a permanent condition of tremor, paraplegia and mutism suggests itself to him, with the result that when the original emotion disappears, its physical expressions remain as hysterical symptoms

In many cases the immediate result of severe emotional strain is stupor As it passes off, sometimes several days later, hysterical symptoms may develop This might occur in casualty clearing stations near the front, in base hospitals, or not until after evacuation to England

In the first two years of the last war cases of this kind were given the unfortunate name of “shell-shock” in the belief that they were organic in origin and the result of actual concussion caused by the explosion of powerful shells Consequently no attempt was made to cure them by psychotherapy, and treatment by rest and sympathy helped to perpetuate the symptoms This was all the more likely to occur owing to the use of the word “shell-shock,” which gave the patient the idea that he was suffering from some new and terrible disease When at last the true nature of the condition was recognised, it was found that psychotherapy not only resulted in the immediate disappearance of the symptoms, when they were treated in the special advanced hospitals opened for the purpose by the British and French and later by the Americans, but cases of two and three years’ standing were also frequently cured at a single sitting in hospitals in England Although this form of hysteria was most common in neurotic individuals, a large proportion of the patients treated within the first forty-eight hours recovered so completely that they were able to return to the fighting line and showed no tendency to relapse Many of those whose condition had persisted for many months before coming under treatment were naturally unable to return to military duty, but such men always became fit enough to go back to their old civil

occupation and often had no underlying mental condition requiring further treatment, although in some cases the hysteria was associated with anxiety symptoms. Indeed, many patients at once lost such symptoms as headache, depression, insomnia and nightmares, which had troubled them for months or even years, directly the obvious physical symptoms, such as mutism or stammering tremor and paraplegia, were removed by explanation, persuasion and re-education.

(2) Hysterical symptoms produced by gassing —The second great group of hysterical symptoms in soldiers resulted from gassing. The irritation of the eyes, throat and stomach caused conjunctivitis, laryngitis and gastritis the latter being the result of swallowing saliva and nasal secretion in which gas was dissolved. The pain caused by the conjunctivitis induced the patient to refrain from opening his eyes with his levator palpebræ superiors if, however he tried to open them, his attempt was frustrated by a reflex protective spasm of his orbicularis palpebrarum. Under ordinary conditions the conjunctivitis had improved sufficiently at the end of three weeks for the eyes to be opened without difficulty, but if the patient was led to fear for his vision on account of previous weakness of the eyes, the previous loss of one eye as in two of our cases, or too prolonged treatment with local applications, bandages, dark spectacles or eye-shades, the voluntary inhibition of the levator might be perpetuated as hysterical ptosis and the reflex spasm of the orbicularis as hysterical blepharospasm. As the uneducated layman associates the idea of blindness with inability to open the eyes, many of these patients thought they were blind. Consequently when they were taught to open their eyes it was found that they could see only indistinctly as they had hysterical paralysis of accommodation, or less frequently they could not see at all, as they had become so convinced that they were blind that they had ceased to look, and, not looking, they could not see. Simple explanation followed by re-education in looking resulted in permanent recovery.

In the same way the whispering in cases of laryngitis, which was originally in part voluntary to avoid pain and in part due to a protective reflex, was frequently perpetuated as hysterical aphonia. This was most commonly the case when an expert laryngoscopic examination had revealed the presence of some abnormal congestion or secretion, which led to intralaryngeal medication, as

both the diagnosis and treatment afforded the necessary suggestion to perpetuate the idea in the patient's mind that his voice was permanently lost. When these patients were taken away from their unfavourable surroundings and treated by explanation, persuasion and re-education, without any recourse to suggestion with the help of anæsthetics or hypnotism or by electricity, they invariably recovered.

The gastritis caused by gassing resulted in vomiting, a protective reflex which fulfilled its object by removing the irritant from the stomach. The actual gastritis rapidly disappeared, and whenever the vomiting persisted for more than three weeks it was always hysterical. A very large number of soldiers were invalided from the service for what was called gastritis, the only symptom of which was vomiting. We found that this hysterical vomiting could invariably be cured by a single conversation, if this was continued until the patient was convinced that he was no longer suffering from gastritis and that he could eat anything without fear of vomiting, even if he had vomited after every meal for many months and had been kept on a strictly fluid diet.

**(3) Hysterical symptoms produced by trivial wounds of limbs**—The most common of all hysterical conditions in soldiers were the paralyses and contractures which followed comparatively trivial wounds of the limbs. A great many different forms were observed, and in many cases the paralysis and contracture were associated with marked vasomotor disturbances and trophic changes in the skin, nails and bones resulting from disuse. The immobility and spasm may arise as a voluntary or reflex response to pain, or they may be due to localised tetanus or to the application of splints or bandages, the abnormal posture assumed and the immobility and spasm being perpetuated by auto-suggestion after the primary cause has disappeared, to which very often is added the hetero-suggestion involved in treatment by electricity and massage when this is not really required. The hysterical paralysis and contracture which result could invariably have been prevented by persuasion and re-education directly the condition of the wound made active movement permissible. Their hysterical nature was proved by their rapid cure with psychotherapy.

**(4) Hysterical symptoms following injury or disease of the nervous system.**—The last group consists of symptoms which are primarily organic and due to an injury or disease of the nervous

system, but which are eventually in part or completely hysterical. When the structural changes produced by an injury or acute disease of the nervous system gradually diminish in extent owing to the disappearance of the vascular and other temporary changes which surround the comparatively small area of total destruction if indeed such an area is present at all the symptoms caused by the throwing out of action of the parts controlled by the nervous tissues primarily involved should disappear *pari passu*. Just as the physical signs in slowly progressive diseases, such as tabes and disseminated sclerosis often precede the onset of symptoms, so in these cases the physical signs are generally still present when the functional capacity has returned to normal, and if the lesion does not disappear completely they may remain as permanent evidence of an organic lesion.

Sometimes however a man does not realise that his functional capacity is improving. If he has been paraplegic he has in the early days made repeated efforts to move his paralysed legs, but without success and he finally gives up the attempt and reconciles himself to the idea of permanent paralysis. If his physician is too much concerned with the possible dangers of early movement he will exaggerate the patient's own fears of permanent disability with the result that the organic paraplegia is gradually replaced by hysterical paraplegia instead of slowly disappearing as the organic lesion becomes more and more reduced in extent. A time may eventually arrive when the paraplegia is entirely hysterical, but, as already pointed out the physical signs of organic disease, such as extensor plantar reflex, ankle-clonus exaggerated deep reflexes and lost abdominal reflex, may still be present. In such cases a diagnosis can be made only by experimental psychotherapy. If as occurred in numerous cases under our care, more or less complete recovery takes place—although of course the permanent physical signs of organic paralysis persist—it is clear that the paralysis was almost entirely hysterical although grafted on an organic basis.

The old method of diagnosing between organic and hysterical paralysis thus breaks down, as the physical signs of organic disease do not, as is too often assumed indicate that the paralysis is entirely organic, but simply that there is an organic element present, which may be quite insignificant in proportion to the hysterical. Moreover it is no help in such cases to consider whether the patient

both the diagnosis and treatment afforded the necessary suggestion to perpetuate the idea in the patient's mind that his voice was permanently lost. When these patients were taken away from their unfavourable surroundings and treated by explanation, persuasion and re-education, without any recourse to suggestion with the help of anæsthetics or hypnotism or by electricity, they invariably recovered.

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the last war, excellent results were obtained. Between 60 and 80 per cent. of men returned to the front line within four weeks of the onset of symptoms. The remainder were sent to the base and of these not more than a quarter were evacuated to England. Only 10 per cent. of admissions into the advanced neurological centres were recurrent cases, and only 2.8 had had more than one recurrence (W. Johnson-Gordon Holmes). The results achieved by the late Col. E. L. Cooper and Capt. A. W. M. Sinclair (1942) during the siege of Tobruk have shown in dramatic fashion how good is the prognosis of war neuroses when properly handled even under the most unfavourable conditions. They worked in a special ward situated in a dimly lit concrete shelter near a heavy anti-aircraft battery where psychotherapeutic talks were punctuated by the noise of shell fire and bombing and where there were no facilities for recreation or occupational therapy. In spite of these disadvantages 79 out of 207 men with war neuroses treated between May and August 1941—i.e. 38 per cent.—were returned direct to their units. An additional 48 went back to full duty after treatment at the base making a total of 61 per cent. now serving as fighting soldiers. Of the remainder 23 per cent. were fit for base duties and only 12 per cent. (of whom 7 per cent. were mental defectives or psychotics) had to be evacuated to Australia.

A large majority of the enormous number of men suffering from war neuroses who eventually reached neurological hospitals in England had never passed through the special advanced neurological centres nor the neurological centres at the base in France. They had been admitted into other hospitals for debility or some other vague diagnosis, and had gradually developed their nervous symptoms through lack of the simple psychotherapy which was necessary to restore their nervous control. Whereas 40 per cent. of cases admitted to the advanced centres had been exposed to high explosive shelling and in some cases actually concussed, the proportion in English hospitals was much smaller. In spite of this there was a tendency to label them all as suffering from shell-shock, even if, as occasionally happened, they had never been out of England.

It is essential for success in treatment that the medical officer should feel convinced that the patient's symptoms are not organic or are at most only in part organic. It is sometimes impossible

is or is not neurotic, as the large majority have no personal or family history of neuroses, and are in every way normal except for the particular symptom from which they are suffering. No more powerful suggestion of hysterical paralysis could be imagined than organic paralysis, and no abnormal degree of suggestibility is necessary for its development

We saw cases of hemiplegia, homonymous hemianopia, which is commonly regarded as always organic, persistent headache, amnesia, and epileptiform convulsions develop after head injuries, paraplegia and persistent incontinence of urine after spinal injuries; paralysis and anæsthesia after nerve injuries in the exact distribution of the nerves, the anæsthesia even resulting in accidental burns, all of which were primarily organic and showed the characteristic features of symptoms caused by an organic lesion, although the recovery with psychotherapy proved that they were hysterical. In many cases, of course, recovery was incomplete, the proportion of hysterical to organic incapacity depending on the extent of permanent damage done to the nervous tissues

### **Hysteria and Wounds.**

Hysterical symptoms of emotional origin were very rare in wounded men in spite of the fact that they had often undergone experiences quite as terrifying as any to which so-called shell-shocked men were exposed. Among 100 patients with hysteria of emotional origin 86 suffered from insomnia and 92 from depression, but neither symptom was present in any of 100 wounded soldiers examined at the same time by Wolfsohn (1918). Moreover, air-raids had a deplorable effect on almost all of the former but did not upset the latter in the least. Although hysterical tremor, mutism and allied symptoms were so rare in the wounded, hysterical contractures and paralysis, which are direct sequels of wounds, were comparatively common. They were purely local manifestations of hysteria and were very rarely associated with any widespread hysterical symptoms or with anxiety neuroses. The probable explanation is that in the confused state, which generally precedes the development of hysterical symptoms of emotional origin, the incapacity and pain caused by a wound absorbs the patient's entire attention so that no hysterical symptom is likely to develop

### **Treatment of Hysterical Symptoms.**

With well-conducted special centres in casualty clearing stations for neuroses, such as were established during the later part of

methods and tried to avoid suggestion altogether whether in the waking or hypnotic state

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Wolfsohn J M (1918) *Lancet* 1 177,

to distinguish with certainty between hysteria and malingering. The patient should then be given the benefit of the doubt, for the distinction is of no great importance, if, when malingering seems possible, the statement is made in the man's hearing that "nervous" cases are cured by the treatment to be adopted, but that "skrimshankers" are not. If malingering is not merely possible but probable, the same statement should be made, but a painful faradic stimulation of the affected part should be added to the treatment.

Whatever treatment is employed, the encouragement produced by the presence of cured patients in the same ward is most helpful. Deaf-mutism is not likely to persist, even if it has been present for many months and many forms of treatment have been tried in vain, when the patient has for a companion a cured deaf-mute, who at once tells him by writing or by the deaf and dumb language how he was quickly cured after being unable to hear or speak for several months. The new patient's mind is prepared for the treatment which he is told he will have on the following day. An "atmosphere of cure," in which the medical officer, the nursing staff, and the recovered patients in the wards all play their part, is of the greatest value for newcomers, however long they may have been ill before admission.

The patient is also made to understand that there is nothing unusual about his case. The extreme interest and sympathy with which he is surrounded account for the frequent persistence of hysterical symptoms for many months spent in a luxurious auxiliary or convalescent hospital. He should be made to realise that many others have had similar symptoms before and have rapidly recovered, and that his early recovery is regarded as a matter of course. The nursing staff should be instructed to speak in the same way, and to tell such patients that they will be perfectly well in a very short time like all their predecessors in the ward.

Hysterical symptoms can invariably be cured by simple psychotherapy in the form of explanation, persuasion and re-education. In the early days of the last war we often used electricity as a form of suggestion, but with increasing experience we found that it was very rarely required. We also at first treated many patients by suggestion under hypnosis and occasionally under partial anaesthesia, but after 1917 we found it unnecessary to use any such

had developed in the trenches. It has followed malaria, cerebro-spinal meningitis trench nephritis, gassing and in several cases muscular rheumatism, which also gave rise to hysterical paralysis of the left arm in another patient. Hysterical paralysis of a leg followed trench fever in one case and paratyphoid arthritis of the hip in another and an officer suffered from hysterical hemiplegia for two years after an attack of sunstroke. In these cases the temporary incapacity caused by the pain or the weakness associated with the primary illness or with some past injury was sufficient to give rise to the idea of paralysis.

*Hysterical monoplegia following trench fever*—A man received a blow on his right knee on August 1st, 1916 from a piece of shell which knocked him down and produced a bruise but the skin was not broken and he was able to carry on. On August 16th he suddenly felt weak and dizzy whilst digging; a severe headache followed and he found he could not walk. This proved to be the onset of a typical attack of trench fever. When the fever had subsided, he found that his right leg was completely paralysed. There were no signs of organic disease. The inability to move the leg was primarily due to a protective reflex, as any movement increased the pain but when the pain had disappeared this inability was perpetuated by auto-suggestion, as it had revived the memory of the injury and he thought himself that the pain was due to the injury. After the psychological origin of the paralysis had been explained to him he was persuaded to walk normally in the course of half an hour.

*Hysterical paraplegia following operation for varicose veins*—A soldier aged 36 was admitted for varicose veins which made his legs ache after marching. The veins were removed by operation on December 22nd, 1942. The patient was allowed to get up on the 26th and was sent to a convalescent hospital on January 7th, 1943. There he found that his legs trembled when he walked upstairs, and in the next few days he became almost completely paraplegic. He was, however, able to move his legs quite normally when lying in bed. He was sent back to the hospital, where no physical signs of organic disease were found. I saw him on January 20th and by simple explanation and persuasion cured his paraplegia so that in ten minutes he was able to walk, run and hop. He was delighted at his recovery and eager to return to duty.

*Hysterical hemiplegia of two years duration and its following sunstroke*.—Lieut. B. aged 24 a well built powerful man, was a farmer before the war. In May 1916 after being a month in Egypt, he had sunstroke. He was unconscious for about three days but was able to leave hospital at the end of three weeks. A few days after returning to duty his horse fell and rolled on to his left leg at the same time he bruised his left shoulder but he was not badly enough hurt to go

## CHAPTER III

### HYSTERICAL PARALYSIS

In this and the following chapters some of the more common war neuroses, which have already been referred to in the discussion on their pathogenesis, are more systematically described

A great variety of hysterical paralyses may occur in soldiers either alone or associated with tremor of the affected limbs. In other cases contractures may be present. The paralysis may be more or less complete, but it often takes the form of *astasia-abasia*, in which movements can be carried out normally or almost normally whilst lying or even sitting, but standing and walking are impossible.

I have already described how temporary organic paralysis caused by cerebral or spinal concussion may be the starting-point of hysterical paralysis. Hysterical paraplegia may also result from the perpetuation of the feeling of weakness in the legs caused by terror.

*Hysterical paraplegia following extreme fear*—Pte C was cut off from his companions during a night raid at Salonica. He thought that he would be tortured if he fell into the hands of the enemy, and found that he could not move as he was "paralysed with fear." He was rescued next morning, but remained paraplegic for three and a half months. He was then admitted into the neurological section at Netley and was cured by vigorous persuasion on the following day.

Severe pain in a limb leads to inability to move it. This is a reflex protective reaction, as movement would be likely to aggravate the injury which caused the pain. The inability to move is partly due to paralysis and partly to spasm of the muscles, which generally regain their normal activity in a short time. But in soldiers, who have become abnormally suggestible from the strain of warfare and who are subconsciously eager for an excuse to leave the Army and return to their homes, the incapacity is likely to become perpetuated by auto-suggestion.

The suggestibility may be so extreme that hysterical symptoms result from auto-suggestion under conditions which rarely if ever lead to them in civil life. Thus I have twice seen hysterical paraplegia develop after an operation for acute appendicitis, which

of the paralysis and with explanation and persuasion quickly cured him. He has now been employed for a year on the ordinary duties of an aircraftsman and is giving complete satisfaction.

### Diagnosis of Hysterical from Organic Paralysis

As hysterical paralysis is a result of auto-suggestion, the paralysis corresponds with the average layman's conception of paralysis. A number of signs occur in organic paralysis, which would not be expected by the uninitiated. They are therefore not observed in hysterical paralysis and serve to differentiate organic from hysterical conditions. In organic hemiplegia, if the supinated arm is completely relaxed and then tossed, it falls in a position of pronation, but in hysteria it does not turn. When a patient with organic hemiplegia grips the observer's hand with his normal hand, an associated movement occurs in the paralysed hand, sometimes even when no voluntary movement can be performed, but no such movement occurs in hysteria. The tendon reflexes of the arm and leg are almost always exaggerated on the affected side in organic hemiplegia, though they may be absent or deficient in the early stages, and ankle-clonus is often obtained in hysteria they are invariably equal on the two sides, and true ankle-clonus, the clonus produced in a completely relaxed limb is rarely present. The abdominal reflex on the paralysed side is weak or absent in organic disease, but equal to the opposite side in hysterical paralysis. The plantar reflex is generally extensor in the former but invariably flexor in the latter (Babinski's sign). When a patient tries to raise himself from the horizontal to the sitting position with his arms folded and his legs widely separated, the paralysed leg rises higher than the other in organic disease but remains on the ground in hysterical hemiplegia (combined flexion of the thigh and pelvis—Babinski's second sign). In a well marked case of organic hemiplegia most or all of these signs are present. In hysteria no single one is present.

In most cases of hysteria the diagnosis is made still more certain by the gait or some other prominent feature being of a nature quite unlike anything seen in organic disease. *Ataxia abasia*, in which the patient can move his legs normally whilst lying or sitting, but cannot stand or walk, is common and is always hysterical. Occasionally some inconsistency about the symptoms points to hysteria. An officer became hemiplegic on the right side after being blown up by a high-explosive shell in the Dardan

to hospital. In the following month he had sunstroke again. The slight injury to his arm and leg now brought the idea of paralysis to his mind, which was in an abnormally suggestible state during the semi-stuporous condition which followed the sunstroke. On leaving hospital three weeks later he returned to duty, feeling fit except for weakness and aching of the left arm and leg. The arm and leg got steadily weaker until they were completely paralysed by January 1916. In June 1917, after a thunderstorm and sitting in the sun he had an hysterical attack, in which he struggled violently and bit everything he could get near to, including the medical officer and his own arm, but he did not bite his tongue. The attack continued with intermissions from 10 p.m. to 2 a.m., when it ceased after an injection of  $\frac{1}{2}$  gr of morphia. He had five more similar attacks. During the fits the muscles of the left arm contracted, but the arm did not move.

On admission to Netley on July 13th, 1917, complete flaccid paralysis of the left arm was present except for some weak movements of the hand. The left leg was very weak and dragged on walking. All reflexes were normal. The prolonged disuse had led to atrophy of the muscles of the left arm, and the hand became blue, cold, and slightly oedematous after hanging by his side most of the day. With persuasion and re-education some improvement occurred in the leg, which he no longer dragged, but he still limped. The fits ceased as a result of hypnotic suggestion, but this had no effect on the paralysis. He was given ether on July 17th. He became very excited, his right arm and leg were held down when he began to struggle, and with the aid of vigorous persuasion and suggestion his left arm and leg soon moved violently. He quickly woke up, and five minutes later he was walking without a limp, and was able to perform every movement of his left arm in a perfectly normal manner. He was still perfectly well in October, 1917, when he returned to full duty.

During the last war I saw several cases of hysterical paralysis in soldiers which had developed as a result of an injury whilst in civil life, but had not prevented a man from getting into the Army. The following is a case in which this occurred in the present war.

*Hysterical dropped wrist in an aircraftsman following a peace-time injury, cured by psychotherapy*—J. R., aged 32, a man of subnormal mental capacity, was employed as a sweeper in an aerodrome as he was unable to do any hard work. Three years before a piece of steel had fallen on his left wrist, since when he had been unable to dorsiflex his left hand. He had received a lump sum in compensation, and in spite of this disability and his lack of intelligence he was earning £8 a week in a munition factory when he was called up for the R.A.F.—In addition to complete paralysis of the dorsiflexion of his left wrist his left hand grip was very weak and there was considerable muscular wasting. Squadron-Leader D. A. Ker recognised the hysterical nature

with sufficient vigour and sufficiently long the first day it was always possible to restore the gait to normal though occasionally as much as two or three hours of continuous treatment might be required. At Seale Hayne Hospital the rapid method was invariably used and prolonged courses of re-education were never required. The day after the patient had been taught to use his limbs normally he was sent to work on the farm, in the workshops or at the pottery. He was given daily physical drill and encouraged to play games of all kinds. In a few weeks he was fit to return to duty or to resume his civil occupation or take up a new one if it was found necessary to discharge him from the Service. The methods we used at Seale Hayne Hospital were soon forgotten and their re-introduction under the designation of rehabilitation is often regarded as an innovation of the present war.

#### Diagnosis of Hysteria from Malingering

The diagnosis of hysteria from malingering is exceedingly difficult as the symptoms are identical. In hysteria they are produced by auto-suggestion and correspond with the patient's own conception of the disability from which he suffers. They are consequently indistinguishable from those which the malingerer voluntarily produces. Hysterical symptoms may disappear on invaliding or receiving compensation, whilst a malingerer may continue to act his symptoms for a time. Though malingering may be suspected in some cases from the patient's mental attitude, it can be diagnosed with certainty only under two conditions. Occasionally an unskilful malingerer may be detected *flagrante delicto*. A momentary movement of a limb when the patient is taken by surprise is compatible with hysterical paralysis, which also disappears during sleep but the appropriate treatment for a paraplegic man, who is discovered walking in the ward when he thinks he is alone and unseen is to send him to the military authorities for punishment. Very rarely a malingerer confesses that he is shamming, but a confession should only be accepted if it is not forced from the man and if it fits in with the facts. This happened with an unwilling conscript who was sent into hospital for supposed epilepsy. Such cases should be sent back to duty at once but without punishment. Pure malingering is very rare in the British Army. Conscious exaggeration of symptoms, conscious prolongation of incapacity which is primarily involuntary and resistance to treatment which is often simply due to obstinacy, apathy or stupidity are compara-

elles He was taken to Cairo and then sent home When he said good-bye to his father, who came to see him at Cairo, he shook him strongly by the hand He told me of this as a remarkable incident when I saw him in London, so it was obvious that he was not malingering He was cured at once by suggestion during hypnotic sleep

### Treatment.

Even now in 1944 it is not invariably recognized that a diagnosis of hysteria carries with it the obligation to cure the patient and that this can almost invariably be done at once and with great rapidity Thus an R A F aircraftsman with hysterical paresis and contractures of his right arm was found to have the medial condyle of his elbow separated from the epicondyle as the result of an injury fifteen years earlier An orthopædic surgeon recommended treating the fracture by grafting The patient was admitted early in 1942 to an orthopædic hospital, where his condition was diagnosed as being entirely "functional" He was then sent back to an R A F. hospital with a note stating "there is very little that can be done for the man," and that "there is no alternative but to board him out" Surely to cure him would have been a more satisfactory alternative! Fortunately this was recognised by Sq-Ldr D A Ker, who proceeded to do so at a single sitting The patient has now gone overseas cured

A paraplegic man is first helped to move his legs whilst he lies in bed, and he is then persuaded to move them without help When he is already able to move his legs whilst lying down, although he cannot stand or walk, he is told that his muscles are now so strong that there can be no doubt he will walk if he summons up sufficient courage to try In both cases without further delay he is made to sit up in bed, stand up and walk, both hands are held for a moment, then one hand, and then his coat is lightly held, and finally he is made to walk alone In most cases the whole process takes less than ten minutes

In long-standing cases the patient assumes a stiff, unsteady gait on learning to walk We assumed at first that this would disappear only after a course of re-education, the patient being made to walk, perform exercises whilst lying and sitting, and swing his legs whilst sitting on a table or holding a chair for a quarter of an hour three times a day We gradually discovered that this was unnecessary if the first treatment was carried out

## CHAPTER IV

### HYSTERICAL CONTRACTURES

Hysterical contractures were extremely common in the last war. Not many medical officers, however, realised that the contractures were hysterical until the end of 1918 with the result that few cases were sent direct to special hospitals for war neuroses. This was due to the fact that a large majority of the contractures followed a wound or other injury to a limb and were not accompanied by any other nervous symptoms. They were generally discovered when the dressings and splints were removed, though occasionally they were noticed directly after the infliction of the wound or they developed gradually several weeks later. They were regarded as a natural result of the injury and were sent to departments for physiotherapy, where they were treated by massage and electricity without any recognition of their true character. In November 1918 Sir Robert Jones visited the Seale Hayne Hospital and saw the rapid cure by psychotherapy of large numbers of cases we had collected from neighbouring hospitals. It was only after this that the hysterical nature of the contractures and the appropriate treatment at last became widely recognised. But unfortunately the modern orthopaedic teaching on the prevention of contractures has not been universally followed, with the result that many cases have already occurred in the present war and their treatment has often been as unsatisfactory as it was in the first years of the last war. According to Watson-Jones (1942) there are still in this country at this moment, hundreds of injured men whose surgical treatment was concluded months ago but whose incapacity is still fatal because minor disease-changes remain, or because their confidence is lacking and moral has been destroyed. Their bodies have been treated but not their minds.

At one time the contractures were thought to be the result of local tetanus. This was the conclusion of the War Office Committee for the Study of Tetanus, and at the discussion on "Muscle Contractures following Injury" at a joint meeting of the Sections of Surgery and Neurology of the Royal Society of Medicine held as late as February 1918 most of the speakers expressed the belief that localised tetanus was the cause in the majority of cases.

tively common. Malingerer may in this way be associated with both organic disease and hysteria. It should also be remembered, as Gilbert Ballet has pointed out, that malingerer may develop into hysteria. a man who pretends to be paralysed for a sufficiently long period may end by genuinely believing he is paralysed.

I saw a man with œdema of a hand produced by a bandage which he had tied round his wrist. He had been four months in hospital, when I first saw him and discovered marks of the bandage at the upper limit of the œdema. Cases of dermatitis artefacta have been seen in soldiers, differing in no way from the same condition observed in civil life. In the last war French and Italian medical officers described cases of "jaundice" caused by picric acid and albuminuria caused by the addition of egg-white to the urine or by cantharides taken by mouth.

Babinaki and Froment's book, illustrates how an implicit belief in their teaching would have led us to neglect psychotherapy, without which the patient could not have been cured.

*Hysterical contracture and paralysis of fifteen months duration at first supposed to be reflex cured by suggestion*—Pte W., aged 21 received a slight wound in the hand in May 1916. When the dressings were removed, his hand was found to be fixed with the fingers semi-flexed and the thumb adducted. Movements of the elbow and wrist were normal, but he could make no movements with the fingers or thumb. The wound soon healed, but the condition of the hand persisted in spite of massage and electricity. It had been supposed that the contracture was due to adhesions involving the tendons and palmar fascia. I first saw him in August 1917 fifteen months after the wound was received. No improvement had occurred, the fingers and thumb being still flexed but it was found that the contracture could be slowly overcome by force. When a voluntary effort was made to move the fingers or thumb the affected muscles were seen to contract, but no movements resulted, as the muscles opposing the desired movement also contracted instead of relaxing. There was no atrophy and the electrical reactions were normal. The hand was swollen—it was red when the room was warm and blue when it was cold, a considerable difference in temperature between the two hands being always present. Excessive sweating also occurred on the palm of the affected hand.

The condition would at once have been diagnosed as hysterical had it not been that a recent study of Babinaki and Froment's work on reflex paralysis and contracture led to the suggestion that this was an example of such a reflex condition, as the position of the hand was identical with that shown in one of the figures in their book, and oedema vasomotor changes, and excessive sweating—which according to them never occur in hysteria but are common in reflex disorders—were present.

As I wished to see how much relaxation occurred under anaesthesia ether was given. The contracture relaxed more slowly than would have been expected in hysteria, this seeming at first to confirm the diagnosis of a reflex condition. But as I still felt doubtful whether the condition might not be hysterical, I tried the effect of vigorous suggestion with the aid of electricity as the patient was coming round. With this treatment the muscles relaxed and all movements became possible. During the next few days treatment by re-education was continued, with the result that ten days later the patient was able to use his hand for all ordinary purposes. Simultaneously with the cure of the contracture and paralysis, the oedema sweating and vasomotor disturbances completely disappeared.

### Pathogenesis

The posture in hysterical contractures is generally that which was assumed immediately after the injury. Thus if one or more

Further confusion arose from the fact that Babinski came to the conclusion that most of the contractures were "reflex" and not hysterical. A monograph he wrote with Froment on "*Hystérie Pithiatisme et Troubles Nerveux d'Ordre Réflexe en Neurologie de Guerre*" was translated into English and the views he expressed, coming from such an eminent authority, were widely accepted. This was most unfortunate, as he concluded that the contractures were not amenable to psychotherapy, but should be treated by physical methods, which led in his experience to very slow recovery after several months.

I was familiar with the condition in civil practice before 1914, and I felt no doubt about its hysterical nature when I first saw cases resulting from war wounds. I was convinced therefore that the contractures were not due to local tetanus and were not "reflex." They were purely hysterical and could be cured very rapidly, generally at a single sitting, by simple psychotherapy. A similar conclusion was reached by Roussy in France and Cohn Russell in Canada. It is well recognised that psychotherapy is effective only when the psychotherapist believes that his method of treatment will be successful. Consequently Babinski, having decided on theoretical grounds that the contractures were reflex and not hysterical, failed to cure them by psychotherapy, although I had received my first lessons in the diagnosis and treatment of hysterical disorders from Babinski himself.

The matter was by no means of merely academic interest. If the contracture is due to localised tetanus, the proper treatment would be by injections of anti-tetanic serum. If the reflex theory of Babinski and Froment is correct, the patients could be very slowly cured only after months of treatment by physical methods. If, however, the contractures are hysterical in origin, they should be curable at a single sitting by psychotherapy.

When hysterical contractures are erroneously diagnosed as being due to localised tetanus or reflex action, the correct treatment will not be undertaken. Thus the popularisation of the idea of reflex symptoms by the publication of Babinski and Froment's book and its translation into English led to hysterical conditions being often diagnosed as reflex, with the result that they did not receive the benefit of psychotherapy, which would otherwise have cured them.

The following case, which was the first one I saw after reading

was fixed in a position of extension by spasm of the sartorius and in two others the knee was flexed and the foot plantar flexed by a localised spasm of a small segment of the hamstrings and the calf muscles respectively. Contractures of this sort could not be imitated voluntarily but the patient had become accustomed to them whilst the tetanus lasted and the habit thus learnt was perpetuated without alteration when the tetanic spasm was gradually replaced by the hysterical contracture.

In many cases the posture is that in which the surgeon fixed the limb by means of splints or bandages when it was first dressed. The patient becomes so accustomed to the immobility of the joint that, when the splint or bandage is removed, he fails to realise that there is nothing to prevent the return of the normal functional activity. He makes a feeble effort to bend the joint finds that it gives rise to pain without any obvious movement resulting and gives up the attempt in despair reconciling himself to the notion that the joint has become fixed as a result of the operation and that no voluntary effort that he can make will have any effect upon it. A little manipulation accompanied by a few words of explanation could at this stage dispel the idea in five minutes, and months of disability would be saved. But too often the after treatment is neglected, or the patient is ordered massage and electricity which are given systematically by sympathetic nurses, with the result that the patient becomes more convinced than ever that there is something serious the matter and that he will get well only with prolonged treatment if, indeed, he will get well at all.

*Hysterical contracture following an operation*—Pte H. aged 30 had the internal semilunar cartilage of his knee removed on February 12th 1918. I was asked to see him on April 5th as he could still walk only with the aid of sticks and was afraid to put any weight on his leg his knee being kept quite stiff. By means of simple persuasion, which took only five minutes he was taught to walk quite normally no limp being perceptible. He walked back to his ward, leaving his sticks behind.

In cases of this kind the posture may alter from time to time if the splint is changed. Thus a man was wounded in the wrist, and his hand was bandaged with the fingers tightly clenched. Some weeks later the surgeon found that the hand had become contracted in this position. Under an anæsthetic the fingers were

peripheral nerves are damaged, the position corresponds with what would result from paralysis, or occasionally from irritation, of these nerves. In such cases, when the nerve recovers from the effect of the injury, which may be within a few hours if the latter is nothing more than slight concussion, or may be weeks or months if it is more serious, the abnormal posture and the inability to move are maintained as a result of suggestion.

In other cases the injury may lead to reflex spasm of the neighbouring muscles and inhibition of movement of the whole limb, which is protective in nature, but which rapidly disappears as the condition of the wound improves. Apart from this reflex action the patient assumes the position which gives most relief to the pain caused by the injury. He keeps the affected segment of the limb rigidly in this position, and at the same time he often abstains from moving the limb as a whole, as this too would cause pain. Both the reflex and semi-conscious local spasm and the general inhibition are liable to be perpetuated by auto-suggestion when the condition of the wound improves and the pain diminishes, the initial reflex and voluntary conditions merging insensibly into hysterical spasm and paralysis.

In some cases the injury itself places the limb in some abnormal position, and this is maintained owing to the pain which the patient experiences when he tries to correct it, and to the reflex and voluntary spasm which oppose the efforts of the surgeon to replace it. This is particularly well seen in the hysterical contracture causing talipes equino-varus after the foot has been twisted by an accident, whether this results in a simple sprain, tearing of ligaments, or a fracture.

Occasionally an hysterical contracture develops as a sequel of local tetanus. In such cases the posture is that caused by the contraction of the muscles, which are supplied by the anterior cornu cells of the spinal cord in the neighbourhood of the point of entry of the afferent nerve fibres connected with the parts immediately adjoining the wound from which the tetanus toxin is absorbed. When at the end of three or four weeks the tetanic spasm gradually disappears, the contracture may persist as an hysterical condition. Apart from the history, this origin should always be suspected when the spasm involves a group of muscles, or the whole or a segment of a single muscle, which could hardly be thrown into action alone by voluntary effort. Thus in one such case the knee

men with no family or personal history of neuroses. They are generally isolated symptoms and are rarely associated with neuroses of emotional origin.

### Ætiology

Hysterical contractures and paralysis may result from injuries to the soft parts of the limb with or without the bones and joints being involved. The commonest cause is a wound of the hand foot, forearm or leg, the symptoms generally developing below and above the injury as well as in its immediate neighbourhood. In most cases no nerve is involved, but in others temporary concussion of one or more peripheral nerves or of the brachial plexus may have occurred or they may have received some actual injury of a recoverable nature. The severity of the symptoms does not vary with the degree of infection or the extent of the injury which is often trivial. They are very rarely associated with severe wounds but I saw several cases in which hysterical contracture and paralysis occurred in the hand after one finger had been amputated, and less frequently in the foot after the amputation of a toe. In other cases a severe injury to one finger resulted in contracture affecting the whole hand. Contractures and paralysis of exactly the same nature developed in the absence of actual wounds—as for example, with fractures, dislocations, sprains and contusions. In a few cases the symptoms followed some minor operation, such as for a ganglion of the wrist, varicose veins in the leg, or an abscess of the arm or leg. In one case it followed plating a fractured tibia three cases followed superficial burns two followed cellulitis and one case of flaccid paralysis of the arm followed anti-smallpox vaccination. Several cases of hysterical contracture and paralysis of one or both legs followed trench foot” and in seven cases the hysterical contracture appeared to be the sequel of localised tetanus.

*Hysterical monoplegia of seven weeks duration following vaccination*  
—Rimm D., aged 21 was admitted on August 12 with paralysis of the left arm. He stated that he was vaccinated on May 30th and on June 28th when at squad drill, he found that his left arm had suddenly become powerless. The limb was hanging useless by his side and the hand and lower 4 inches of the forearm were blue and cold, the palm being mottled and purple. Persuasion for a quarter of an hour produced movements of the arm in half an hour the colour and temperature were normal and all movements of the limb including the fingers

now fixed on a splint in a position of extreme hyperextension. Six months later his hand was still rigidly fixed in the new position. He came under the care of Captain A. Robin, who rapidly cured him by psychotherapy. As the posture is artificially produced by the use of splints, it may sometimes be one which cannot be imitated voluntarily, but it is none the less hysterical, the patient having been trained to maintain the position whilst the limb was fixed.

In all these conditions the development of the hysterical contracture and associated paralysis is due to the fact that the patient fails to realise that there is no reason why the spasm should not relax and the power of movement return, when the primary factor—nerve injury, protective reflex, conscious or subconscious antalgic spasm and inhibition of movement, localised tetanus, or fixation by splints or bandages—is no longer operative. The patient regards the contracture and inability to move as a direct result of his injury, and naturally ignores the intermediate cause, such as the pain or tetanus. If it had been pointed out to him when the pain was disappearing that his incapacity was due to the pain and only indirectly to the injury, and that there was therefore no longer any reason why it should be maintained, he would have made the necessary effort and the hysterical condition would never have developed. But not having been told this, and perhaps having been ordered treatment with massage or electricity at a stage when such treatment could have been of no real use, as recovery from any organic injury which may have been present at first was now more or less complete, the idea of incapacity was confirmed in his mind, and the original auto-suggestion was fortified by the unconscious hetero-suggestion of the doctor and the masseuse, with the result that the contracture and paralysis were perpetuated as hysterical conditions after the primary cause had disappeared.

From what has been said above, it is clear that contractures are always associated with more or less paralysis, but the reverse is not the case, as uncomplicated hysterical flaccid paralysis is not uncommon. In many cases the paralysis is more extensive than the contracture, a contracture of the hand, for example, being sometimes accompanied by paralysis of the whole arm. Whereas most war neuroses occur particularly in nervous individuals—the martial misfits—contractures occur with equal frequency in normal



FIG. 1.—Hysterical "main d'accoucheur" following wound of forearm.

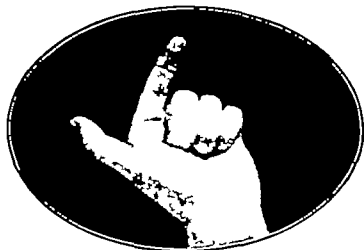


FIG. 2.—Hysterical contracture of hand persisting thirty five months after wound near elbow (Capt. C. H. Ripman.)



FIG. 3.—Same hand as Fig. 2 after half an hour's treatment, showing depressions formed in palm by pressure of nails.

were perfect after three quarters of an hour, though the hand-grip was not perfect until the next day (Captain G McGregor)

*Hysterical "main d'accoucheur" and anæsthesia following concussion of median nerve by gunshot wound of the forearm*—Sergt M was wounded in the right forearm on April 10th, 1917, a compound fracture of the middle third of the radius being produced. When the splints were removed three weeks later, his hand showed a typical *main d'accoucheur* deformity, the fingers being rigidly extended and pressed together. Total anæsthesia was present in the area supplied by the median nerve. As no improvement had occurred by November, an operation was performed, and the tendons and median nerve were freed from adhesions. This, however, aggravated the contracture, so he was transferred to us on December 21st, 1917.

Well-marked vasomotor disturbances, excessive sweating, and considerable atrophy of the soft tissues and nails were present. On the day of admission the hand was continuously but painlessly manipulated, the patient being persuaded at the same time that it would rapidly relax. In ten minutes complete relaxation was obtained and the deformity had disappeared. The fingers, which had not been moved voluntarily for eight months, could now move normally, but an area of total anæsthesia was still present. The anæsthesia was doubtless originally organic in origin, but as it now extended considerably beyond the area supplied by the median nerve, we thought that it must be hysterical. Suggestion with the aid of faradism for a few minutes resulted in its complete disappearance. With the return of movement the vasomotor and secretory disturbances disappeared. The patient was kept under observation for three weeks, and as there was no incapacity he then returned to duty.

### Variety of Postures.

In most cases a single segment of a limb is involved, the hand and foot being most frequently concerned. Very often, however, the whole or greater part of the limb is affected, but in such cases the contracture and paralysis are most severe in the immediate neighbourhood of the injury, although in rare instances the whole limb is completely paralysed and rigid.

#### *Upper Limb*

Hysterical contracture and paralysis of the hand give rise to three common types of posture. The fingers may be extended, in which case they are generally also adducted towards the middle finger, a *main d'accoucheur* resulting (Fig 1), in one case the fingers were abducted as well as extended. The other extreme is for the fingers to be clenched tightly into the palm of the hand. All the fingers may be involved, or only two or three, in which case one finger

fingers are affected, but sometimes especially when the condition has followed an injury of the hand or wrist, only those fingers most directly involved by the wound are implicated, the others being either normal or comparatively slightly affected (Fig 4)



FIG 6.—Hysterical contracture of toes persisting for nine months. (Capt. S. H. Wilkinson.)

Occasionally the fingers are in a position of extreme hyperextension, which may be so marked that it suggests that the condition cannot be hysterical in origin. In most cases of this sort, however it is



FIG 7.—Same case as Fig. 6, cured after half an hour's treatment.

found that the patient has always been able to hyperextend his fingers, and a similar though rarely so well marked, hyperextension can be produced by voluntary action in the other hand.

may occupy an intermediate position. In these cases the skin often becomes sodden with sweat owing to the impossibility of evaporation and the difficulty in washing. In one case, in which the position had been maintained for two and a half years, the nails of three



FIG 4 —Hysterical contractures of three fingers persisting for nine months after wound of wrist, cured within an hour by psychotherapy

fingers were found to have produced depressions about a quarter of an inch deep into the palm of the hand (Figs 2 and 3). In the third variety the hand takes up an intermediate position very similar to that assumed normally at rest. In the extended and



FIG 5 —Hysterical dropped wrist following wound of wrist, improved in an hour and cured in three days after persisting for twenty-four months

clenched position the paralysis is always accompanied by more or less contracture. The intermediate position is that generally assumed in flaccid paralysis, but it may also be associated with contracture, though this is rarely severe. In most cases all the

but no improvement resulted. At Christmas 1917 he was sent to a convalescent hospital from which he was transferred to us on February 14th 1918. He stammered badly and had severe clonic movements involving both legs, which increased with voluntary movement so that he was unable to stand and could only walk a few unsteady steps with the aid of sticks when further progress was prevented by the violence of the clonus. As no signs of organic disease were found I told him that the treatment he would have the following day would cure him and the presence in his ward of three men who had recently been cured of paraplegia helped to convince him that this was true. By 5 p.m.



FIG. 2.—Hysterical talipes equino-varus following a sprain, cured in half-an-hour after persisting for nine months.

the next day as a result of persuasion and re-education, he could talk slowly without any stammer and he walked a quarter of a mile with a gait which was normal except for a lump due to left-sided talipes equino-varus.

As I could not at first overcome the deformity of his left foot by manipulation, I thought it might be due to some organic injury especially as an operation had been performed upon it after a radiographic examination in London. Moreover it was colder than the right foot and was always wet with sweat, whilst the right foot was dry evidence which would be accepted by Babinski as excluding hysteria and suggesting a reflex origin. More vigorous manipulation, however two days later continued for an hour and a half, resulted in complete disappearance of the deformity and the patient was now

When the wrist is involved it is commonly in an intermediate position or flexed. In rare cases it may be flexed to the extent found in complete organic musculo-spiral paralysis (Fig 5)

The hand is frequently held in a position midway between supination and pronation. When the contracture is incomplete, it is almost always possible to obtain greater movement in the direction of pronation than supination, inability to supinate the hand completely being very common.

When the elbow is involved, it is almost invariably flexed, but I have seen two cases of extreme extension. The flexion is generally such as would correspond with the position of the arm when held in a sling.

When the shoulder is affected, the arm is generally held rigidly to the side. In incomplete cases, and when recovery is taking place spontaneously, it is often found that the elbow can be raised to the level of the shoulder, but it is impossible to raise it any further.

### *Lower Limb*

In rare cases the toes alone are affected. One patient, who had been wounded in the thigh, developed a persistent flexor contracture of his toes, which disappeared as a result of psychotherapy after it had persisted for nine months (Figs 6 and 7).

In the commonest variety of hysterical contracture and paralysis involving the leg the foot is extended in a position of talipes equinus. This is often associated with varus, which may be present alone. In some cases an extreme degree of talipes equino-varus is present. These postures may be due to simple flaccid paralysis, but there is generally some degree of rigidity, and in many cases the contracture is so extreme that not the slightest movements can be obtained by ordinary means (Fig 8).

*Hysterical rigid talipes with paraplegia, tremor, and stammer of fourteen months' duration, cured in two days*—Sergt-Major P, aged 32, was blown up in December 1916, and remained unconscious for some days. He was paraplegic, stammered badly, and had a generalised tremor when admitted to a provincial hospital, where he remained for three months. He was then transferred to a hospital in London, where he remained for nine months. He was treated with massage and electricity, but very little improvement occurred. A piece of bone was removed from the left foot in October in order to correct some deformity,

well. At the end of four days he was put on horseman's work and physical drill as he walked quite normally except for a slight tendency to allow his knee to turn inwards (Captain S. H. Wilkinson.)

### Changes Secondary to Deficient Circulation

The normal circulation through a limb depends upon its active movements. If for any reason the arm is not moved in cold weather the hand becomes shrivelled, white or blue, numb painful and stiff. These well-recognised changes disappear at once with active exercise and on warming the limb both of which restore the circulation. The tendency to disturbances of this kind is much greater in people with a poor circulation than in those with a naturally good circulation. The former class know by experience that they must keep their fingers constantly moving in cold weather in order to avoid getting numb.

It is thus natural for the immobility caused by paralysis or contracture of a limb whether organic or hysterical, to result in deficient circulation and the usual secondary changes. As however the paralysis is continuous, whereas the physiological inactivity referred to lasts only for a few minutes or at most for a few hours at a time, the secondary results are likely to become much more profound especially in individuals with a poor circulation. Such deficient circulation is generally congenital, but in soldiers it is often acquired or aggravated as a result of exposure. Thus in all cases in which circulatory complications are severe the circulation in the normal hand is also feeble. The limb especially its peripheral part, becomes cold and white or blue and the diminished blood supply results in a diminution in the volume of the hand, except in the cases in which cedema develops. Pressure on the skin with a finger is followed by a very slow return of blood to the part rendered anæmic.

When the venous and lymphatic stasis is very marked and the paralysis absolutely complete, cedema may occur especially if the paralysis is accompanied by contracture in a position in which the veins and lymphatics are obstructed by the rigid muscles. The cedema is sometimes very considerable, and may give rise to the impression that there has been some injury of the blood vessels (Fig 9). This, however is not the case, for with the return of voluntary movement the cedema disappears in the course of a few days as the circulation becomes once again normal.

More or less stiffness of the joints is generally present in cases

able to walk without any limp. It is obvious, therefore, that the talipes, like the paraplegia and stammer, was hysterical in origin.

The knee is frequently involved, extension and flexion being about equally common. Extreme flexion is rare, but rigid fixation in a position of semi-flexion is often seen. Contractures in the position of extension are frequently so extreme that not the slightest movement can be obtained actively or passively until relaxation is produced by psychotherapy.

*Hysterical stiff joint following gunshot wound of the knee, cured by persuasion*—Sergt W, aged 23, sustained a gunshot wound on the outer side of his left knee on January 16th, 1918, which penetrated the joint. At the casualty clearing station the foreign body was removed, the joint was washed out, and the capsule closed. He reached a hospital in Scotland on January 30th, and on February 20th he was allowed to get up, but the limb was still in a splint and he had to use crutches. Four months later, according to his documents, adhesions of the knee-joint were broken down under an anæsthetic, and he was sent to a command dépôt. He was admitted to Seale Hayne Hospital on August 20th, walking with the aid of a stick with a very bad limp owing to inability to bend his left knee. Except for the scar of the incision on the outer side of the joint the knee appeared normal, but he could only flex it to about 120 degrees. The stick was taken away, and with persuasion and re-education for half an hour complete flexion was obtained, and he walked with only a slight limp. The next day he was able to go up and downstairs and walk on the level quite normally. (Captain G McGregor)

When the hip is involved, the limb is generally in a position of slight flexion, but rigid extension is sometimes seen. Adduction or abduction and eversion or inversion may be present.

*Hysterical flaccid foot-drop, with inversion of the leg, following a gunshot wound of the right thigh a year before, rapid recovery with persuasion*—L-Cpl F, aged 23, was wounded by shrapnel in the outer side of the thigh on May 11th, 1917. A counter-opening was made and a piece of metal removed. He began to get about on crutches in August 1917, but always with his foot dropped and his leg turned inwards. On his medical case-sheet a specialist reported "there was some involvement of the right sciatic nerve." He was transferred to us on May 11th, 1918, with a spring attached to his boot to keep his foot up, and he could only get about with difficulty with the aid of sticks. It was explained to him that, having held his leg so long turned inwards and with his foot down, he had forgotten how to use his muscles. With simple persuasion and re-education he quickly learnt to walk quite

apparent tearing down of adhesions and the production of inflammatory reaction do not necessarily mean that the condition is organic.

By measurement with a sphygmomanometer, Babinski and Froment found that the amplitude of the pulsation of the arteries in the affected limb is greatly diminished compared with that on the normal side. They found that this inequality disappears on warming, and Roussy and his colleagues (1919) using exactly the same methods, noted its disappearance when the paralysis and contracture were cured by psychotherapy proving that it is simply a result of the deficient activity of the limb.

The long-continued deficient circulation in hysterical paralysis also leads to malnutrition. Atrophy of the subcutaneous tissue occurs, with the result that the volume of the limb particularly the tips of the fingers greatly diminishes, and the increased blood-supply which occurs on warming the hand and on recovery from the paralysis produces only a comparatively slight increase in size, most of the loss of volume being due to actual atrophy.

This is well shown in Fig 10 which represents the first finger of a paralysed hand compared with that of the sound hand. The normal lines on the hands especially the transverse lines on the dorsal aspects of the joints of the fingers, become less marked and may even disappear. Owing to the diminished bulk of the fingers, the skin becomes too large for its contents and is thrown into longitudinal folds, especially over their palmar aspect. The muscles atrophy for the same reason, and also as a result of simple disuse as their normal metabolism depends upon their activity. It is well known that the corresponding anterior cornu cells of the spinal cord atrophy from disuse after a limb has been amputated. It is probable that this also occurs in hysterical paralysis, as the disuse may be just as complete.

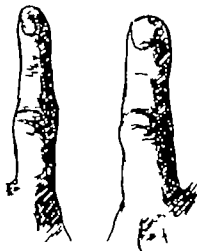


FIG 10.—Atrophy of index finger of left hand, compared with normal finger of right hand, resulting from hysterical contracture of left hand of eighteen months duration. Drawn after recovery from the contracture.

of long standing, even in the absence of any muscular spasm. This may be so extreme that not the slightest movement is possible. It is most obvious in the joints of the hand, particularly at the metacarpo-phalangeal articulations. In such cases so-called adhesions are often broken down under an anæsthetic, actual tearing being heard, and this may be followed by effusion into the joint. This has not unnaturally been regarded as evidence of organic change, but in all hysterical cases, and, I believe, in some cases of organic paralysis, the stiffness is not really due to any permanent organic alteration in the tissues. A considerable amount of relaxation occurs when the circulation is improved by immersion in hot water. In hysterical cases, even of many months' duration, directly the patient has been taught to contract his paralysed muscles and to relax any spasm which is present, the return of movement so improves the circulation that often



FIG 9—Hysterical contracture of right hand of fourteen months' duration, associated with severe œdema, cured in two hours (Capt A W Gill)

in the course of a few minutes the condition of the joints and surrounding tissues returns to normal. This condition is probably the result of some coagulation process in the fibrous tissue caused by the accumulation of products of metabolism, which are normally removed by the blood when the circulation is efficient. Improvement with warmth and recovery with psychotherapy must be due to the improved circulation resulting in the removal of such products permitting the temporarily coagulated fibrous tissue to assume its normal fluid consistence.

As the stiffness is due to circulatory disturbances and not to any muscular spasm, whether hysterical in origin or not, it naturally persists under an anæsthetic, and when the joint is forcibly moved, the surrounding structures which are torn are not abnormal fibrous tissue, but simply the normal periarticular tissue, which is temporarily in an abnormal condition owing to the deficient circulation. The injury to the normal tissues results in effusion. This is a point of the greatest importance, as it follows that the

the paralysis and contracture disappear with psychotherapy they disappear with them. The return of movement is always accompanied by an improvement in the circulation, and the limb regains its normal colour and warmth. The numbness and stiffness of the joints, however closely the latter may have simulated an organic condition completely disappear. As soon as the limb is used as much as it was formerly—which should occur immediately the first treatment is concluded—and the circulation is consequently restored, the supply of nourishment becomes normal again and at the same time the active contractions of the muscles stimulate their metabolism, so that they together with the soft parts, the bones and the nails, gradually return to their original condition.

Fig 12 shows very strikingly the change in the nutrition of the nail which follows recovery. The drawing was made about six weeks after the patient had been cured by psychotherapy from

hysterical paralysis which had lasted for over a year. The new nail, which had grown since the cure of the paralysis, was in every way normal. The distal part of the nail, which was white, rough and thin, in sharp contrast with the pink, smooth and thicker proximal portion, is what was left of the nail present before recovery took place. Three weeks later the last trace of the abnormal nail had been replaced by healthy nail.

#### Diagnosis

The diagnosis of an hysterical contracture depends primarily upon the incompatibility of the symptoms with the injury. On careful examination it is generally at once clear that the exact form and extent of the contracture and paralysis cannot possibly be explained by the injury inflicted upon the affected muscles or their nerve supply. In some cases an injury to a nerve appears to be responsible for some of the paralysis present, but it is often found that the paralysis extends to muscles supplied by other nerves which could not have been injured. Moreover an injury to a nerve cannot account for persistent spasm of the muscles it supplies and still less for that of

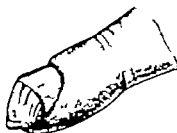


FIG 12.—Improved nutrition of nail, drawn six weeks after sudden recovery with psychotherapy from hysterical paralysis, which had lasted for over a year (Capt. A. W. GILL)

The x-rays show abnormal transparency of the bones, which is apparently due to deficient calcification, but we have never observed any alteration in their outline (Fig 11, Plate I) Lastly, the nails become thin, brittle and abnormally opaque, and they often show longitudinal grooves

Babinski and Froment rightly pointed out that the phenomena I have just described as accompanying functional paralyses and contractures caused by minor injuries to limbs were not hysterical, because they were not due to suggestion and were, indeed, incapable of being suggested, and because they could not be removed by direct psychotherapy Unfortunately Babinski and Froment went further, and concluded that the paralyses and contractures, which were associated with these non-hysterical phenomena, were therefore also non-hysterical and must be due to the same cause as the latter Although they were still uncertain as to their origin, they thought that the most likely explanation was that both the paralysis and contracture and the associated phenomena were reflex in origin, and corresponded with the atrophy and contracture in the neighbourhood of diseased joints, which Vulpian and Charcot long ago described as being due to reflex action

We were, however, convinced that this explanation was incorrect, and that the paralysis and contracture were hysterical, and the associated phenomena simply secondary to disuse Cases of contracture, in which all the signs described by Babinski and Froment were present, occurred in the absence of any wound, so that a reflex origin was excluded, their hysterical nature being subsequently proved by the cure which followed psychotherapy Babinski and Froment had found considerable difficulty in curing paralyses and contractures of this nature by psychotherapy, which confirmed their opinion that they could not be hysterical But our experience was more fortunate, and we did not see a single case in which psychotherapy failed to produce complete recovery We had cases precisely similar to every one of those of which photographs were reproduced in their book on "*Hystérie-Pithiatisme et Troubles Nerveux d'Ordre Réflexe en Neurologie de Guerre*" The paralyses and contractures exactly fulfil the requirements necessary for a diagnosis of hysteria, being produced by suggestion and being curable by psychotherapy The associated phenomena do not fulfil these requirements and are therefore not hysterical But they are none the less the direct result of the hysterical symptoms, for when

the paralysis and contracture disappear with psychotherapy they disappear with them. The return of movement is always accompanied by an improvement in the circulation and the limb regains its normal colour and warmth. The numbness and stiffness of the joints, however closely the latter may have simulated an organic condition, completely disappear. As soon as the limb is used as much as it was formerly—which should occur immediately the first treatment is concluded—and the circulation is consequently restored the supply of nourishment becomes normal again and at the same time the active contractions of the muscles stimulate their metabolism so that they together with the soft parts, the bones and the nails, gradually return to their original condition.

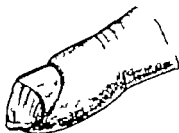


FIG. 12.—Improved nutrition of nail, drawn six weeks after sudden recovery with psychotherapy from hysterical paralysis, which had lasted for over a year (Capt. A. W. GILL.)

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other muscles. Persistent muscular spasm resulting from peripheral injuries is thus invariably hysterical. When a part of the condition is obviously hysterical, this should be cured, and an attempt made to cure the residual condition, however closely it may simulate the results of an organic lesion. For the latter is often perpetuated as hysteria, and recovery of function may take place before the disappearance of the accompanying physical signs of organic disease, such as extreme muscular atrophy, loss of reaction to faradism, and abolition of the deep reflexes. I have already pointed out that vasomotor and trophic changes, which have in the past been regarded as conclusive evidence of organic disease, may be the result of disuse from hysterical paralysis, and that it is most important to recognise how impossible it is to distinguish by simple manipulation the stiffness of joints due to organic changes in the surrounding tissue from that due to local deficient circulation, quite apart from any associated muscular spasm. For the diagnosis from localised tetanus, *vide* p 417

### Prophylaxis.

If the modern teaching of British orthopædic surgeons were adopted there would be no hysterical contractures to treat. In a statement issued on November 2nd, 1940, by the British Orthopædic Association and its Standing Committee on Fractures it is laid down that "active exercise and rehabilitation must begin on the day that active surgical treatment begins—that is, on the first day. Some joints may need to be immobilised, but every other joint is actively exercised, and functional activity is thereby maintained even in the immobilised region." The statement was a reply to an editorial in a medical journal, in which it was said that "there are three stages in the treatment of an injury. First comes the active surgical treatment with some period of immobilisation immediately after, then a stage in which massage and passive movement are the chief remedial features, lastly the stage of active exercise and graduated work." Such a view is, as the orthopædic authorities wrote, "a complete contradiction of the principle of modern fracture treatment, which represents a survival of the obsolete practice of immobilising injured bones, and then after several weeks or months discovering for the first time that joints have become stiff." This applies equally well to all injuries to limbs, including those in which no bone or joint is involved. Watson-Jones (1942) has recently published a descrip-

tion of the admirable rehabilitation service which has been available for the Royal Air Force since 1940. According to Balne (1942) in the great majority of hospitals the bad old methods are still in use. Limbs are subjected to prolonged immobilisation and even if massage and exercises are prescribed no attempt is made to provide daily medical supervision of such remedial measures or to fill the patient's day with a programme of planned exercises, occupational therapy and organised games directed towards the prevention of the disability with which he is threatened. As a result barely a week passes without the transfer to rehabilitation centres of the same dreary list of the products of excessive immobilisation—stiff backs, painful wrists and shoulders, flexion deformity of knees and that most ungainly progeny of fractured tibia out of persistent plaster—namely, plaster equinus.

#### Treatment

In the last war we had almost entirely given up hypnotism in the treatment of hysterical conditions by the end of 1917 as our results were uncertain, and we had seen so many patients in whom prolonged treatment by hypnosis before admission had proved unavailing and had even given rise to new symptoms. We rarely used suggestion with the aid of electricity except at the commencement of treatment in some cases of flaccid paralysis, in which the contractions produced in the paralysed muscles by faradism powerfully appeal to the patient's mind.

Our final method was to begin with a full explanation of the cause of the symptoms in language suited to the patient's intelligence and degree of education, followed by persuasion and re-education combined in most cases with manipulation, which doubtless acts to some extent by suggestion. A very important, but by no means essential, preliminary is the creation of a proper atmosphere of cure. Directly the patient is admitted, the sister encourages him to believe that he will be cured as soon as the doctor has time to see him and she is often helped by the spontaneous efforts of other patients in the ward, who have been rapidly cured of similar symptoms and tell him of their cure. The medical officer sees him some hours later and after examining him and coming to the conclusion that the condition is hysterical, he tells him as a matter of course that he will be cured the next day. By the following morning the patient is fully convinced that the hoped for cure will take place. As the medical officer is equally convinced that he will cure the

patient, the two essentials for recovery are present. The nature of the actual treatment is really immaterial, but simple explanation, persuasion and re-education have the great advantage of making the patient take an active part in his own cure and remove any suspicion of charlatanism from the proceedings

The patient is made to understand that any treatment he has already received has prepared the way, so that nothing now remains but a properly directed effort on his part for complete recovery to take place. He is made to watch the contraction of the muscles and the play of the tendons of the normal limb, and to attempt to imitate them in the affected one

In some cases it is not even necessary to touch the patient, mere explanation and persuasion being sufficient to cause him to relax any spasm which may be present, and then to perform the various movements of the part with quickly increasing strength and rapidity. In other cases the limb requires to be lightly supported at first, but here again no passive movements are employed. In cases of this kind the patient often expresses astonishment at the simplicity of the treatment, which succeeds in curing him in a few minutes, in spite of the fact that he has previously been given massage and electricity for months without any obvious result. The explanation is that now for the first time he is made to take an active part in the treatment—to use his will-power, instead of allowing himself to become a passive agent in the hands of the masseuse. A few physiotherapists realised the importance of active effort. But I frequently saw a patient reading a newspaper which he held with one hand, whilst his other hand was being massaged or treated with electricity, exactly as if it did not belong to him. I very rarely saw a patient being encouraged throughout his massage or electrical treatment to take an active part in the proceedings. As a matter of fact, neither electricity nor massage is of any use in these cases, although the massage and electrotherapeutic department of military hospitals and command depôts were crowded with them. Recovery should take place very rapidly, and then nothing but the ordinary activity of everyday life is required to restore any residual weakness due to the muscular atrophy caused by prolonged disuse.

In many cases of severe contracture more active manipulation is required. In our earlier cases we often forcibly moved the limb in the direction opposed to the normal action of the contracted

muscles, however much the patient might complain of pain the movements being repeated until complete relaxation and the power to perform voluntary movements were restored. We subsequently realised that the production of pain often produced a more or less subconscious resistance to the treatment. We found that in every case it was possible by explanation and persuasion to get the patient to relax his muscles, so that passive movements could be carried out with the production of little or no pain, however powerful the spasm might be. When little or no resistance to passive movements persists, they should be combined with and finally replaced by active movements until the condition of the limb is normal.

When very great difficulty is experienced in getting the contracted muscles to relax, the limb should be placed in hot water and the manipulations carried out when the circulation has been artificially improved in this way. Some relaxation always occurs, because, as I have already pointed out, the rigidity is in part the direct result of the deficient blood-supply.

Passive movements are most effective if carried out by the medical officer himself as the patient is engaged in conversation the whole time and made to take an active part in the movements from a very early stage in the first sitting. For this reason we never employed the mechanical appliances for performing passive movements, which became very popular during the last war. Even when complete relaxation of spasm has been attained and the normal movements have been restored to a hand, the patient is very apt to forget this, and to continue to make the other hand do the work of both. After the condition has persisted for many months or even three or four years, it is only natural that this should be the case at first if he does not think about it, as he has become so unaccustomed to having two useful hands. It is, therefore of the greatest importance to give the patient some occupation, such as basket-making or toy making, which will necessitate his using both hands from the very day on which recovery occurs. At Seale Hayne Hospital we found that the men enjoyed working at a pottery we instituted better than anything else, and this had the desired effect of rapidly training them to use the hand which had previously been paralysed. At the same time the patient is instructed to be careful to use both hands for all ordinary purposes, such as dressing and feeding himself, and in the case of the right

hand he should at once begin to write with it again. A musician should be encouraged to play his instrument.

Finally, the patient should be taught to keep the limb in the proper position by an effort of will, in order to restore the normal postural tone and postural length of the muscles. When the leg has been affected, it is necessary to give some re-education in walking for a few minutes in order to overcome any tendency to limp, due to the persistence of the abnormal postural length of the muscles involved in the contracture and of those which acted abnormally in order to compensate for the partial disability. Here again we never found any apparatus, such as Zander's, necessary for the re-education of normal movements and restoration of full capacity. The ordinary activity of everyday life is quite sufficient, if the patient is made to understand that he must now use his limb exactly as if he had never been injured.

The following case is a single example out of a very large number, showing how rapidly complete recovery occurs, even in very long-standing cases in which all hope of cure had been given up.

*Hysterical paralysis of the left deltoid of twenty months' duration, and hysterical contracture of the right biceps and triceps of nine months' duration, cured in a few minutes*—Pte C, aged 28, was wounded superficially over the left shoulder and right elbow in August 1916. He was sent to England and was in five different hospitals. He was transferred to Seale Hayne Hospital on May 14th, 1918, with a diagnosis of "spasm of the right biceps". His right elbow was rigid and flexed at slightly more than a right angle. He was unable to abduct his left arm, the deltoid being quite flaccid. The latter condition had evidently been given up as hopeless, as no mention of the paralysed left shoulder was made on his medical case-sheet. He had twice had his right arm forcibly straightened under an anæsthetic in other hospitals, but on each occasion the spasm had returned. With explanation, manipulation and persuasion the resistance of the muscles of the right arm was overcome in a few minutes and he was taught to move his left arm in every direction. He was immediately put on farm work and physical drill, and both arms soon became quite strong. He was discharged to duty on July 10th, 1918. (Captain S. H. Wilkinson.)

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## CHAPTER V

### RHEUMATISM, SCIATICA, AND HYSTERICAL POSTURES AND GAITS

An abnormal posture and gait are frequently assumed by men suffering from back ache following minor injuries and fibrositis and from the various conditions which give rise to sciatica. The posture and gait are those which have been instinctively found to cause least pain and to make walking as easy as possible in spite of any weakness which may be present. The injury or disease may be quite trivial, and the posture and gait often persist long after all pain and weakness have disappeared. The patient says he limps because of pain the truth is that he has pain because he limps. The attitude which gave relief when the original painful condition was present now causes pain, because muscles which are normally little used are called into continuous activity and quickly become fatigued while ligaments and other muscles are stretched. The abnormal postures generally disappear on lying down, although in the pre-hysterical stage when the posture is protective they are maintained in all positions. Such cases may be very resistant to treatment but a combination of explanation persuasion and re-education eventually leads to recovery.

#### Sciatica

Heaven help the unsuspecting individual who wanders into orthopedic out patients these days and admits having sciatica. (Anonymous surgeon, quoted by Campbell Golding 1939)

By sciatica is meant pain in the distribution of the sciatic nerve. It was formerly thought to be almost invariably caused by inflammation of the perineural sheath and interstitial tissue of the sciatic nerve except for a small number of cases of referred pain from disease of the hip joint. Later inflammation of the nerve roots was regarded as equally common. Many neurologists still believe neuritis and radiculitis to be the most frequent cause, though the vision of an inflamed and swollen sciatic nerve has never yet been granted to human eyes (Symonds, 1943). Since Love of the Mayo Clinic in 1939 described how protrusion of the nucleus pulposus of the fourth and fifth lumbar intervertebral disc causes sciatica by exerting pressure on the fifth lumbar or first sacral

nerve root, all neuro-surgeons and some neurologists consider this to be a common condition and true neuritis and radiculitis to be rare Symonds (1942), for example, recently stated that his experience with Service patients has convinced him that this is by far the most frequent cause of sciatica But according to Good (1942) the pain in the vast majority of cases both in soldiers and civilians is referred from disease of the quadratus lumborum, glutei and tensor fasciæ latæ muscles, it very rarely arises in the roots or trunks of the sciatic nerve An Army Medical Bulletin published in October 1942 also states that fibrositis is the commonest cause of sciatica

The most recent theory concerning the pathogenesis of sciatica is that of an ex-president of the British Orthopædic Association, who has expressed his conviction that spinal arthritis is by far the commonest cause (Bankart, 1943) Like Good, he does not even mention intervertebral disc prolapse as a possible alternative He believes that the inflammation spreads from the joints to the spinal nerves in the intervertebral foramina and so causes neuritis In his experience most cases respond to heat, massage and exercises with or without manipulation, but persistent or recurrent pain calls for removal of the lateral intervertebral joints, an operation which "rarely, if ever, fails to cure the sciatica"

It is difficult to reconcile these divergent views, but a review of some of the many forms of treatments, which have in turn been popular during the last fifty years, suggests the likely explanation

### The Treatment of Sciatica . 1901-43

"For immediate relief the deep injection of morphia over the nerve is the most effectual treatment Not infrequently we see suffering which has been almost intolerable thus removed, as by magic, within a few minutes" (Fagge and Pye-Smith, 1901) After mentioning this treatment and also the injection of chloroform into the nerve, Osler (1912) adds "it is remarkable how promptly, in some cases, the injection of distilled water will relieve the pain" Osler also advised a trial of acupuncture "the needle should be thrust deeply into the most painful spot for a distance of about two inches, and left for from fifteen to twenty minutes" This treatment is still sometimes used Walshe (1941) writes that "acupuncture of the nerve with a series of specially designed needles is a useful and ancient remedy which acts by puncturing

the sheath of the nerve and allowing the escape of inflammatory exudation

A popular treatment between 1920 and 1930 was injection into the nerve trunk of 50 to 80 c.cm. normal saline solution the effect of this, according to Wilfred Harris (1926) was to cause it to swell up in an egg-shaped form separating the nerve bundles and bursting asunder laterally any adhesions that have been formed. Soon it became apparent that equally good results were obtained when the nerve root was massaged and the saline solution was injected somewhere in its neighbourhood when there could be no question of breaking down adhesions.

I do not know who first suggested substituting oxygen for saline solution but this treatment was widely practised for a few years. Kinnier Wilson in his *Neurology* (1940) ascribed the benefit which followed to the provision of a sort of air cushion for the inflamed nerve a quaint variation from the earlier notions about breaking down adhesions.

In 1930 William Evans reported the results of treating forty cases of 'primary idiopathic, essential sciatica' by the intra-sacral injection of about 80 c.cm. of fluid into the epidural space. As normal saline solution was as effective as novocaine and as pain was often experienced in the course of the nerve during the injection, it was presumed that it acted by stretching the nerve roots and this was found to occur by experiment on the cadaver. Complete relief was obtained in 60 per cent. of cases and considerable benefit in 13 per cent. For a time this method was widely used, but as it was more difficult to carry out without being any more effective it was gradually replaced to a great extent by the older methods of injecting in or near the nerve trunk.

Good (1942) believing that the origin of the pain in sciatica is almost always muscular states that he obtained permanent relief almost instantaneously by injecting procaine into each myalgic area in all but one of sixty five cases, the one exception being not unnaturally assumed to be a malingerer. In striking contrast with the permanent results claimed by Good, Hyndman, Steinder and Wolkin, who were apparently unaware of his work, published results which seem to prove that his success must have been the result of suggestion and of nothing else. Out of the fog of ignorance which has hung for so long over the subject they wrote it has become clear that all cases are due either to direct pressure by

a herniated intervertebral disc or to a reflex neuralgia from a small, well localised painful area in the fibrous tissue of the lower part of the back. Apparently every case of disc pressure was subjected to operation, as there is no mention of any alternative treatment, and the possibility of spontaneous recovery with rest is not mentioned. In the fibrositic cases injection of procaine into the tender focus completely abolished both local and radiating pain. "*The patient was warned that with the wearing off of the procaine effect in from several hours to several days the pain, both local and radiating, would return worse than ever. Having enjoyed a brief spell of complete relief, he keenly resented the return of his complaint.*" Treatment by immobilisation (traction, plaster, brace, cast and occasionally operative), hot packs, massage, cathartics and aspirin then led to gradual recovery.

Soldiers are even more easily suggestible than ordinary people, so that when they are sent to "the man who cures sciatica" they have their minds well prepared for his particular form of suggestion therapy. As Good finds myalgic points in 100 per cent of soldiers with sciatica it is obvious that he must produce them by unconscious suggestion in at least 50 per cent—a minimum estimate of the proportion caused by pressure on the roots or inflammation of the trunk of the sciatic nerve. Having suggested a myalgic point it is immaterial how he suggests it away, and an injection of novocaine is as good a method as any other, although no doubt an injection of morphia, saline solution, air or nothing at all would do equally well so long as there is a prick, as with the older forms of unconscious suggestion therapy by injection into or near the trunk or roots of the sciatic nerve. It is inconceivable that the analgesic effect of novocaine could have any direct action, because the anæsthesia it produces is very evanescent, as Hyndman and his colleagues' patients found to their cost, and it could not influence the inflammation or other changes in the tissues which are supposed to produce the myalgic spots. The whole story is reminiscent of Babinski's method of curing hysterical paralysis. He had proved by a long series of investigations that hysterical anæsthesia is always the result of unconscious suggestion by the observer, and had no difficulty in inducing cutaneous anæsthesia in the paralysed limb by gross suggestion. He then cured the hysterical anæsthesia by painful faradism, at the same time suggesting that the associated paralysis would disappear, which it invariably did.

Hugh Wingfield, who had a great reputation for his success as a hypnotist about twenty five years ago invented a treatment of sciatica, a description of which was published in 1917 by Sainsbury as Wingfield was too modest to do so himself. The treatment consisted of painting the skin over the course of the sciatic nerve with fuming hydrochloric acid, the skin was left uncovered till the fluid had completely evaporated. Although it would burn a hole in linen, it produced little or no irritation of the skin, so did not act by counter irritation. Wingfield obtained uniformly satisfactory results, even in the most chronic cases, and Sainsbury was also much impressed with its value. I used it a number of times myself and there was no doubt that it often led to the rapid disappearance of the pain. The fumes were so unpleasant that one had to protect one's eyes and nose during the application, and I have little doubt that the psychological effect of having the fuming fluid applied to the painful area was the cause of the success of the treatment.

### The Rational Treatment of Sciatica

Civilians engaged in hard manual labour and soldiers ought to be particularly easy to cure because they are unable to carry on with their duties from the first day of the attack and so seek medical help at the earliest possible moment just when treatment by rest gives the best results. Rest in bed from the onset of symptoms leads to rapid improvement and often to complete recovery in two three or four weeks. Complete immobilisation by splint or plaster is not often required as the patient keeps sufficiently quiet and is far more comfortable if allowed to choose his position and vary it slightly from time to time. But in severe cases, as I first recommended thirty years ago immobilisation by means of a plaster spica should be carried out. In Symonds's (1943) experience this is more successful in such cases than any other treatment. The patient should throughout be encouraged to expect rapid recovery sufficiently complete to allow a return to full duty within a fortnight of getting up. It will then be unnecessary to employ any of the forms of suggestion hitherto in use— injection into the nerve by the neurologist, the intervertebral space by the expert or as near the nerve as he can get by the less expert, into the myalgic spots by those who believe in them, manipulation or a plaster case by two rival schools of orthopaedic surgeons, removal of

herniated intervertebral disc by the neuro-surgeon and of the lateral intervertebral joints by Bankhart, or the application of fuming hydrochloric acid by the magician. Complete rest should be maintained until there has been no spontaneous pain for five days. The patient should then be given active movements in bed and should be allowed up a day or two later. At the same time any abnormality in his posture on standing and in his gait on walking should be promptly corrected by explanation, persuasion and re-education. With such treatment very few patients will be unable to return to full duty within two months from the onset of symptoms. Without the simple psychotherapy involved in promoting the expectation of rapid recovery followed by rapid rehabilitation there is a great tendency for the pain and disability with the abnormal gait and posture to be perpetuated and exaggerated as an hysterical symptom. This is especially likely to occur in the Services, where a man may subconsciously see in his illness a way of escape from an uncongenial life to the comparative comfort and safety of home, and among civilian manual workers, who subconsciously see in it a holiday and possible financial benefit. Hysterical sciatica, with the associated hysterical posture and gait, may last for weeks or months until it is ultimately cured by the gross suggestion, unwittingly given by the believers in the various forms of treatment I have already discussed, helped in the soldier by discharge from the Army and in the civilian worker by a successful claim for compensation. It can also be cured by simple psychotherapy in the form of explanation, persuasion and re-education when its hysterical nature is recognised—often with great rapidity, even in very long-standing cases, as we found in the many cases of the kind in soldiers which we treated at Seale Hayne Hospital during the last war.

It is important to remember that a diminished or lost ankle-jerk and wasting may persist long after the complete disappearance of active disease. Their presence does not therefore indicate that persistent pain is due to organic disease, although it shows that organic disease must have at one time been present.

The few patients who do not get well after a month of complete rest in bed are among those who give a history and show physical signs of root pressure. A further period of rest may still lead to recovery, but if there has been little or no improvement they should be referred to a neuro-surgeon, who will cure them by removal of the herniated intervertebral disc. Pennybacker (1942, 1943), who has

verified the lesion at operation in about 150 cases in the past three and a half years, believes that the clinical syndrome of prolapsed disc is sufficiently characteristic for a diagnosis to be made without recourse to lipiodol injections which may set up a troublesome irritant reaction in the theca. All cases are probably traumatic in origin, but the injury may be trivial and no more than a strain. This is followed by low back pain often diagnosed as lumbago which may be severe or nothing more than slight discomfort. The incident is often forgotten and not related by the patient to the subsequent sciatica, and, as Penhybacker points out even in cases in which the sciatica follows closely on the lumbago the memory of the latter may be swamped in the more severe existing sciatica pain. Symonds is convinced that so far as Service patients are concerned operation, even in the best hands, has proved so unsuccessful in getting men back to duty that a prolonged period of immobility should be advised, and when this fails to render a man fit for duty within a reasonable time he should be invalided. On the other hand from my limited experience I have no doubt that in severe cases, in which complete rest has failed to cure the results of operation are remarkable as the patient obtains immediate relief from pain and is soon able to return to full activity.

### The Bent Back of Soldiers

A soldier walking slowly and painfully with the aid of two sticks, bending far forward, his arms and legs often shaking with the effort, was a common sight in the latter part of the last war. Though a large literature grew up around him in France, where authors vied with each other in inventing new names for him, such as *plicature vertébrale camptocormie* (αδύρρω I fold) (Souques and Mlle Rosanoff-Saloff) *campto-rachis* (Laignel Lavastine and Courbon) and *spondylosse antalgique* (Sicard) it received comparatively little attention in England. This might have been regarded as a matter for congratulation were it not for the fact that the true nature of the condition was rarely recognised, with the result that many men who should have been cured in a few minutes with psychotherapy drifted from one hospital to another were treated with electricity and massage, spent many weeks undergoing hydrotherapy at various spas, and ended by being invalided from the Army as totally incapacitated and convinced from the failure of treatment whilst in the Army that their incapacity would be permanent. Considerable experience convinced me that

the bent back of soldiers, however variable its ætiology, was constant in its pathology, being always hysterical in nature and consequently always curable by psychotherapy (Hurst, 1918)

### Ætiology.

The most common cause was a blow on the back, from the explosion of a shell hurling a man against the side of a trench or causing the parapet to fall on him, or from some accident which might equally well have occurred in civil life, such as being jammed against a wall by a lorry. Occasionally a wound was present, but this was always superficial, the spine itself having escaped. A history of "muscular rheumatism" was obtained almost as frequently, pain in the back having followed exposure to cold and damp, generally in the trenches, but occasionally at the base or even in English camps. In a typical case seen in February 1941, the condition had developed soon after an attack of mild gonococcal arthritis of one ankle. The rigid bent back had been present for three months and was thought to be the result of ankylosing spondylitis, possibly of gonococcal origin. The day after its hysterical nature was recognised the patient was persuaded to move it freely and a few days later he bicycled fifteen miles. At the end of a fortnight he returned to duty. Photographs of a typical case were shown at the 1941 meeting of the Association of British Physicians as an example of "pseudo-spondylitis ankylopoietica". No abnormality had been found to account for the posture, and it was assumed that it was a result of lesions resulting from nitrogen emboli formed whilst working in caissons at high pressure. It was added that there was no evidence of any hysterical factor—but the posture itself was sufficient evidence of hysteria. Several of our patients appeared to have been predisposed by having always walked with a slight stoop or having had previous attacks of lumbago. The father of a boy I saw at Lemnos was a "martyr to rheumatism" and walked with a bent back, but this probably predisposed him more by the mental association than by any inherited physical condition.

### Pathogenesis.

The one factor common to all cases is pain in the back, which is relieved by bending far forward (*spondylose antalgique*). The patient finds he can stand and walk with some degree of comfort only in this position, and at first he generally lies in bed curled on his side. After a time the pain caused by the blow on the

back the rheumatism or other exciting cause diminishes or disappears. The patient can now often rest comfortably lying flat on his back, but he continues to stand or walk in the same bent position. He does not realise that this posture was originally adopted voluntarily in order to relieve his pain and he regards both the posture and the pain as direct and independent results of the injury or disease which they followed. The pain has gone, but the posture adopted with the object of relieving it remains. Unfortunately the medical officer frequently makes the same mistake as the patient and prescribes treatment with diathermy radiant heat whirlpool baths massage or other elaborate method in some institute for physiotherapy with the result that the notion of serious disease is still further impressed upon the patient's mind, the original auto-suggestion being thus reinforced by hetero-suggestion. How little benefit follows such treatment unless it is given as a form of suggestion is proved by the fact that the Bath physicians, with whom I discussed the question in 1918, told me that they did not know of a single soldier with a bent back who was cured by treatment at Bath.

Numerous investigations with the x rays showed that there was no organic spinal lesion, even in those cases which followed a severe blow on the back. In several cases the radiologist had reported a "doubtful" spinal deformity fracture, or other pathological condition, which further examination and the course of the disease proved to be non-existent. But the patient often saw the report, with the result that the difficulties in the way of successful psychotherapy were much increased.

The bruising which follows an injury and the fibrositis which is the basis of so-called muscular rheumatism, myalgia, and lumbago are true organic lesions, but they quickly disappear and the condition which persists for weeks or months afterwards is entirely hysterical.

The patient generally complains of more or less pain in his back but, though very prone to exaggerate his symptoms, he is generally ready to admit that the pain is less severe than it was at the onset of his illness. Whatever part of it is genuine is probably the result of constantly stretching the small muscles and ligaments of the spine, which in normal individuals is bent for a few minutes at the most instead of for the greater part of each day. That this is true is shown by the immediate improve-

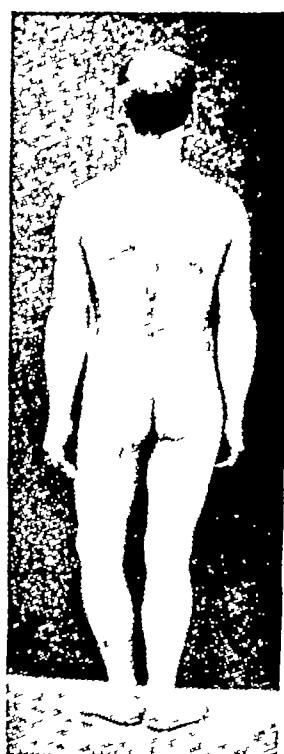
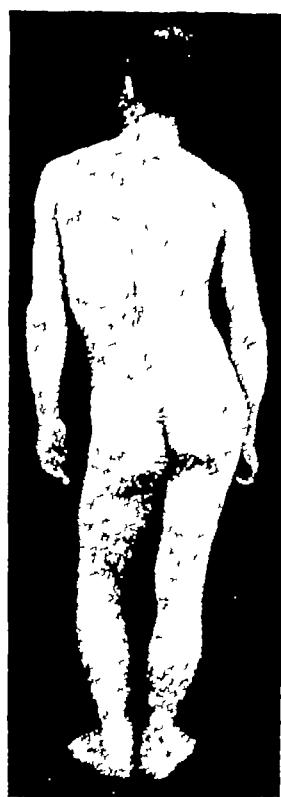


FIG 13 —Case of hysterical spinal curvature, *top* before treatment, *bottom* after treatment

ment or disappearance of the pain when the patient is taught to assume a correct attitude

### Symptoms and Diagnosis

The appearance of the patient is so characteristic that the diagnosis is at once obvious, as there is no organic condition which in any way resembles it except senile osteo-arthritis of the spine, which never affects men of military age. Moreover the position assumed in the latter is the same whether the patient lies down or stands whereas the bent back of soldiers almost invariably disappears entirely or almost entirely on lying down. In one case I was able to persuade the patient whilst lying supine to arch his back sufficiently to make it possible for him to support himself on his heels and head although he could only stand and walk with his back rigidly bent far forward. Other diseases of the vertebrae are excluded by the absence of tenderness over the spine though the muscles may be sensitive to pressure. French writers described in detail the folds which are observed in the skin of the abdomen and back of the neck, but these do not differ in any way from those which develop when a normal individual acts the part of a very old man—bending forward and throwing his head back so that he can look straight ahead

In the large majority of cases uncomplicated kyphosis is present, but we also saw cases of lordosis scoliosis, and of combined kyphosis and scoliosis of similar origin.

*Combined kyphosis and scoliosis of twenty months duration cured by re-education in an hour and a half*—Pte D., aged 30 with over two years service was digging a trench in April 1917 when he felt something give in his back. Pain developed and his back became bent. He carried on until June 1917 when he was partly buried by a shell. He was sent to the base where he remained until July when he was transferred to England for discharge. On admission there was a lateral curvature of the spine to the left in the dorsal region and to the right in the lumbar region with corresponding prominence of the left shoulder and right hip. Very marked lordosis was also present (Fig 13 (top)). He swayed violently backwards and forwards when standing with his feet together. The faulty position disappeared on lying down and was easily corrected by manipulation on standing. After an hour and a half's re-education he had no further difficulty in keeping his spine in a position free from curvature both when standing and walking (Fig 13 (bottom)) and was discharged from hospital a fortnight later (Captain G McGregor)

In one case, in which recovery followed treatment by persuasion

under Captain C H Ripman, the kyphosis was associated with curvature in a horizontal plane, the shoulders being thrown far forward, an appearance of severe pigeon-breast resulting

*Hysterical kyphosis associated with curvature in a horizontal plane* — Pte B was wounded on March 21st, 1918. On admission in August he had a superficial scar, three inches long, running diagonally across his back opposite the third dorsal vertebra. He showed extreme kyphosis, which produced two deep horizontal folds across his epigastrium, below which the abdomen was round and prominent. His chest was also hollow from side to side, with very marked prominence of the points of his shoulders. He breathed very badly, with no abdominal movement and very little expansion of the lower part of his thorax. He looked so pigeon-breasted that the deformity had been regarded as organic, though he himself said none was present until he was wounded. The curvature did not disappear on lying down. With persuasion his body was gradually straightened out, his shoulders being pressed back until both the kyphosis and the side-to-side curvature, together with the hollowing of his chest, had disappeared. After prolonged persuasion he was able to move freely, and the depression of his epigastrium and the prominence of his abdomen with the horizontal furrows across his epigastrium disappeared. Three weeks later, being quite fit, he was sent back to duty.

In the following case, under Lieut S H Wilkinson, lordosis instead of kyphosis followed burial. The patient's posture and gait closely simulated that of pseudo-hypertrophic muscular dystrophy.

*A case of pseudo-pseudo-hypertrophic paralysis cured by persuasion after lasting for ten months* — Rfmn P, aged 20, had had six months' active service in France, when he was buried by a shell in September 1917. He was not dug out until four hours had elapsed. He felt none the worse for it, except for a slight pain in the back, but he was sent to hospital the following day with a diagnosis of trench feet, his feet being very blue and cold. He was kept in bed for a month and then transferred as a cot case to a hospital in England, where he remained for two months. He was kept in bed the whole time, the only treatment given being for his feet. He was then sent to an auxiliary hospital. At the end of a week he was allowed out of bed for the first time, when he found that he could walk only with difficulty. In spite of treatment with electricity and massage his gait became more and more difficult. He was transferred to Seale Hayne Hospital on July 15, 1918. On admission he presented many of the characteristics of pseudo-hypertrophic muscular paralysis. His gait was waddling, very marked lordosis was present, he was unable to bend forward, sit or lie down without help, and if he lay flat on his back he was unable to get up. As there was no atrophy of any of his muscles and no family

history of paralysis it was recognised that the condition was hysterical. After continuous treatment for two hours by persuasion he had greatly improved, and with further persuasion during the following three days he completely recovered.

In some cases, especially when the exciting cause is a blow on the back, the gait is stiff each step requiring a great effort, and a generalised tremor may be present, the patient sweats excessively and has an anxious and haggard expression.

### Treatment

All forms of treatment except psychotherapy are useless. Thus electricity unless employed as a means of suggestion, massage, and fixation in a plaster jacket after straightening the spine under an anæsthetic, as recommended by certain French writers can do only harm by fixing the idea of disease still more firmly in the patient's mind.

Any form of psychotherapy is likely to be efficacious. I hypnotised my first patient, a nineteen year-old soldier whose back had been bent for two months after rheumatism contracted at Gallipoli. He walked quite erect whilst hypnotised, and I woke him up whilst he was still walking. Though it had been arranged for him to embark for England the next day he asked to be allowed to stay at Lemnos, as he was now cured, and a week later he returned to the Peninsula at his own request. In spite of this success we did not again employ hypnotism in such cases, as we preferred to use simpler methods, which were just as effective. We explained to the patient how the position he assumed on standing and walking was simply a bad habit, contracted when the originally severe pain in his back could only be relieved by bending forward. He was made to realise that the fact that he could lie down with his back straight proved that there was nothing serious the matter with his spine, and that he would in fact lose such pain as he had *on standing* if he were to relax his muscles and stand erect. He stood with his back to a wall with his heels touching it, and his shoulders were then gently but firmly and persistently pushed back till they also touched the wall. He was told that the pain he felt whilst this was being done would disappear directly he was erect and the less resistance he offered the quicker he would be cured. In most cases relaxation rapidly followed and in a few minutes he found he could stand erect without support. He was then taught to walk in the same posture and recovery was complete.

A discharged soldier was cured in this way in a quarter of an hour without admitting him to hospital, he was to return to his home in Australia a few days later, and the condition had existed for eleven months in spite of all kinds of treatment

In the following exceptional case simple psychotherapy failed, but a trick resulted in a dramatic recovery.

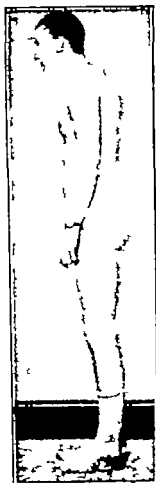
A man, aged 31, who had always stooped slightly and had suffered from lumbago for twelve years, developed a very severe degree of kyphosis (Fig 14 (a)) as a result of falling out of a buggy whilst home on leave. He was treated by massage, blistering, and immobilisation for two and a half months and then by thermal baths and massage for two months at Bath without any benefit. After this he was admitted to Seale Hayne Hospital, but resisted our ordinary methods of treatment by persuasion and re-education for five weeks. He refused to believe that his condition was curable, and disciplinary measures only rendered him more resistant to treatment. I therefore had a board made with a foot-piece fixed at right angles to its lower end. He lay flat on the board with his feet on the foot-piece without difficulty, as he had all along been able to lie down with his spine fully extended. The upper end of the board was then very slowly raised, the lower end resting on the floor (Fig 14 (b)). He was encouraged to remain lying on the board with his muscles relaxed and his arms lying loosely by his sides. The upper end of the board was raised higher and higher until at length the board was perpendicular and the foot-piece was resting on the floor. As he did not alter his position in relation to the board, he was now standing quite erect, so was told to walk off the foot-piece, which he proceeded to do without bending his back (Fig 14 (c)), and he was delighted to find that at last he was cured. There was no relapse, and the cure of his physical condition was immediately followed by a complete change in his mental outlook. Instead of being morose, depressed and disagreeable, he became cheerful, happy and grateful for all that had been done for him.

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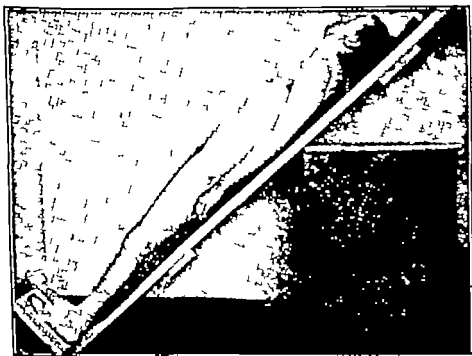
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(a) Before treatment.



(c) After treatment.



(b) During treatment.

Went back of six months duration cured by "back board method."

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## CHAPTER VI

### HYSTERICAL TREMOR

Hysterical tremor is among the commonest of war neuroses. It is generally coarse in character and may simulate the tremor of paralysis agitans or disseminated sclerosis. It may affect the whole body or be limited to the head, jaw one side of the body, both arms or both legs, or a single limb. It is generally increased when any effort is made, and an "intention tremor" may develop owing to the patient's fear of missing an object he is told to touch. It may follow severe emotional strain, whether the patient has been actually blown up by the explosion of a shell or not. It is often associated with an expression of terror and other emotional manifestations, such as excessive sweating, tachycardia, and vasomotor instability. The emotional factor is well seen by the constant aggravation of the tremor by excitement and by sudden noises, such as the slamming of a door or thunder which remind the patient of gun-fire. An air raid always causes an acute exacerbation of symptoms.

In the first three years of the war of 1914-18 the tremor was generally regarded as due to some organic change in the central nervous system caused by emotional shock and by concussion when it followed the explosion of a shell in the immediate neighbourhood. Psychotherapy completely failed in the hands of those like Meigs in France and Oppenheim in Germany who held these views, and they therefore believed the prognosis was very serious.

At first, like Meigs, we regarded psychotherapy as almost without influence on tremor. But we later found that in the majority of cases it was as amenable to treatment as any other hysterical symptoms even if it had persisted for many months.

Tremor is always associated with more or less rigidity of the affected part, a condition of *faislé trémulante* resulting. We found that directly this rigidity was overcome by persuasion and re-education the tremor disappeared, just as it disappears when relaxation occurs during sleep or under deep hypnosis. By explaining this to the patient and at the same time gently rubbing the affected muscles, and then performing passive movements and tossing the

limb or the head and rolling it from side to side, complete relaxation is gradually produced, the tremor disappearing *pari passu*. In many cases of severe tremor of the head both the rigidity and the tremor can be caused to disappear in a few minutes, it rarely persists for more than an hour if the treatment is continued. In severe cases associated with great exhaustion it is wise to keep the patient quietly in bed for two or three days and to give him ten grains of bromide three times a day before commencing active treatment. But he should be promised, even at this early stage, that as soon as he has had a short rest steps will be taken to cure him. There is often a tendency to a slight recurrence with excitement, but this can be easily controlled, and complete recovery nearly always takes place within a week. We watched numerous cases for weeks or even months after their recovery, they were able to do hard work on the farm without any return of symptoms. One patient with very severe side-to-side tremor of the head, who was cured in this way, drove the hospital motor-car for three months without the slightest sign of relapse, and another was employed as barber.

Tremor in soldiers, being thus curable by persuasion and re-education, must clearly be hysterical. At the onset it represents one of the physical results of painful emotions, but when it continues after the emotion has disappeared it is purely hysterical.

Although primarily the emotion gives rise to the tremor, there is no doubt that the persistence of tremor helps to perpetuate the patient's anxiety neurosis and renders him miserable, lachrymose, and abnormally easily frightened. This is proved by the fact that the cure of the tremor immediately results in the expression of terror and depression disappearing from the patient's face. At the same time his outlook on life becomes completely altered. In severe cases, in which a history is unobtainable from the patient owing to the severity of the tremor and the associated stammering, which are greatly aggravated directly he is spoken to, the cure of the tremor and stammering results in the patient being able to give a good account of himself without any emotional accompaniment. From a considerable experience of both methods we concluded that better results were obtained by attacking the physical accompaniment of the emotions first, instead of trying to deal with the emotional factor whilst the tremor was still present. In numerous cases the cure of the physical symptoms resulted in immediate

recovery from very trying anxiety symptoms, which thus required no direct treatment at all.

The hysterical stammer which frequently accompanies hysterical tremor is due to a similar *fixité tremulante* of the muscles concerned in speech, and can be rapidly cured by similar means (*vide* p 69)

## REFERENCE

Meige H (1916) *Rev Neur*, 33, 201

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this he slept badly had occasional fits, and remained very nervous. He was sent home for "debility" in September 1916 and was discharged to duty in December. He had six fits in the first week after returning to France three in hospital there two on the boat, and between two and four on each of the first four days after admission. He had never passed urine or bitten his tongue in a fit but the description sounded like true epilepsy and this was the diagnosis made by a M O in France who had seen him have one. As at that time I had not seen a single case of epilepsy resulting from the war without actual injury or a personal or family history of the disease I doubted the diagnosis. I therefore hypnotised him and suggested he would have a fit he at once had one and though very similar to true epilepsy the plantar reflexes remained flexor instead of extensor. I told him this would be his last fit and he had no more except on February 16th and 17th when I talked to him about returning to duty. This was in striking contrast to the absence of any effect with bromide, which he had in France and during the first four days after admission.

### Diagnosis

The diagnosis of hysterical from true epileptic fits has generally to be made from the patient's own account of his attacks. A history of involuntary micturition and much less frequently involuntary defecation, or of injury to the tongue caused by biting, are the only definite points suggesting true epilepsy which can be obtained from the patient himself, and these are inconclusive except when a visible injury to the tongue is present. A man rarely if ever has an hysterical fit when he is by himself but he may have one during sleep as a result of a vivid nightmare. If his companions say that he has struggled with them or clutched at objects near him, the fits are certainly hysterical, but in many cases it is impossible to communicate with those who have seen him in a fit. Even when the medical officer is fortunate enough to be present during one, it is much less easy to come to a definite conclusion than is generally supposed. I have seen several cases, in which a diagnosis of true epilepsy made under these circumstances before admission has been proved by subsequent events to be erroneous. The only signs which can be regarded as conclusive evidence against hysteria are definite cyanosis, complete loss of conjunctival and corneal reflexes, loss of the pupillar reaction to light, and, most conclusive of all, an extensor plantar reflex. The plantar reflex should be tested in every case as soon as the convulsions have ceased and before consciousness has returned. Unfortunately a flexor reflex does not definitely exclude true epilepsy

## CHAPTER VII

### HYSTERICAL FITS

#### **Ætiology.**

Hysterical fits are much more common in soldiers than might have been expected from their rarity among men in civil life

The idea of convulsions may be already present in the man's mind, if he has previously suffered from true epilepsy or if he has been the witness of convulsions in some near relation, horror or fright may then suggest an attack. Such a history may arouse the suspicion that the fits are genuinely epileptic and not the result of suggestion, as strong emotions may be the exciting cause of a fit in epileptics. But if a description of the fit is obtainable, or if subsequent spontaneous or induced attacks are witnessed, their hysterical nature becomes clear

*Hysterical fits in a man who had had convulsions as a child*—A New Zealand soldier was rendered unconscious for a few minutes by concussion from a high-explosive shell. He had had a few fits after falling on his head when eight years old, and the subconscious recollection of this probably led to the occurrence of hysterical convulsions at least once and generally several times a day. He was subsequently hypnotised and recovery was suggested, with the result that the fits immediately ceased and did not recur

*Hysterical fits following horror suggested by seeing a man with convulsions*—An officer and his servant were blown up by a shell. The servant did not lose consciousness and ran to fetch a stretcher. On his return, the officer, to whom he was greatly attached, made a few convulsive movements and then died. He immediately had a fit, and in the following two months he had eleven more. They ceased completely after their origin and nature had been explained to him

In rare cases there is no apparent reason why convulsions rather than some other hysterical symptom should develop

*Hysterical fits reproduced and cured by hypnotic suggestion*—Pte R, aged 27, a typical "martial misfit," who had been a professional singer and subsequently a valet in private life, joined the Army in July 1915. There was no family history of epilepsy, and he had never had a fit himself. Soon afterwards he was sent to France, where he worked in a canteen. A week later some men broke in at night, and a mallet was thrown at him, although he was not hit he immediately had a fit and remained dazed, dumb, and unable to walk for two days. After

been proved and their repetition contra-suggested in this way though the bromide was discontinued. On one occasion I wished to demonstrate the production of an hysterical fit in a man, who had already been told under hypnosis that he would have no more. Although the first fit had been produced with the greatest ease, the suggestion of cure had been so successful that all attempts to produce a fit during hypnosis now failed completely. I was unable to produce a fit by suggestion in another patient, but the hysterical nature of his convulsions was proved by the fact that they completely ceased after the suggestion of cure which I had made during hypnosis.

#### EPILEPSY IN SOLDIERS

True epilepsy may develop as a result of the strain of active service but I saw only three undoubted cases in which the individual was not predisposed by having previously suffered from major or minor epilepsy or by having a family history of epilepsy except when an actual head injury was the exciting cause. In my experience almost every exception proved to be apparent as further observation showed that the fits were really hysterical or were the first manifestation of some gross organic nervous disease, such as general paralysis.

Many men who had inherited a predisposition to epilepsy but had never suffered from it, or who had had no fits since early childhood, developed epilepsy when exposed to the strain of active service. In some cases an acute infection and in others concussion by exposure to blast was the exciting cause.

Automatism and amnesia were exceptionally common manifestations of epilepsy in soldiers. Men, who had previously suffered only from ordinary major epilepsy or petit mal, and often only at rare intervals or not since childhood, were found wandering far from their units and were unable to give any account of themselves. They ran grave risk of being punished for desertion unless the true nature of their condition was recognized. It is clear that no man who has ever suffered from epilepsy should be admitted into the Army.

though an extensor reflex is much more commonly present and may persist for five to fifteen minutes after the convulsions have ceased. In all cases of doubt, when there is no personal or family history of epilepsy, and when one has to rely entirely on the patient's unsupported statements, I have tried to induce an attack by suggestion under hypnosis. In hysteria a fit is generally produced at once, its characteristics can be studied at leisure, and it can be made to last as long as desired. I do not think that true epilepsy could be induced in this way, and I have certainly seen no case in which a fit produced by suggestion was not definitely hysterical and in which the plantar reflex was not flexor.

In rare cases soldiers sham fits. As the convulsions, like those of hysteria, have the character which the individual himself regards as most natural, fits due to malingerer and hysteria are clinically quite indistinguishable. It is therefore only the special circumstances of the case which may lead to a suspicion of malingerer, and definite proof is not obtained except in rare instances, such as in the following case, in which the man admitted he was shamming.

*Malingerer of epilepsy by an unwilling conscript*—Pte C., an unwilling conscript, had numerous fits for the first time in his life on board ship coming from Jersey three days after being called up, and had fifty more in the two days he was in hospital at Southampton. He was then sent to Netley. As the history pointed to the fits being either hysterical or more probably due to malingerer, I hypnotised him and suggested a fit. Nothing happened. I then said to the Sister in the patient's hearing that it was quite clear he was shamming, as in all genuine cases a fit would result from this treatment. He immediately had a fit with marked opisthotonos, which was abruptly terminated when I ordered him to stop and wake up. I then told him I was sure he was malingerer, and that he would be wise to admit it. He did so at once, and promised that he would not have any more.

### Treatment.

I have already described the manner in which hysterical fits can be diagnosed from true epilepsy by inducing one by suggestion under hypnosis whenever the patient's history is unreliable and no fits have been observed by the physician himself. This method of diagnosis can be combined with treatment by telling the patient before, during, and after the fit that this one will be the last he will ever have. The treatment proved most successful. Many men, who had been having numerous fits every day in spite of large doses of bromide, had no more after their hysterical nature had

was kept in bed and reading was forbidden for fourteen days, then high frequency daily sparks from my throat, and I was given chloroform, but with no effect."

I told him that he would be cured with the treatment he would have the next day and a man who had been rapidly cured after being dumb for a year was instructed to tell him about his recovery. By the next morning he felt convinced he was going to get well. I passed a catheter down his larynx, and after a few seconds of vigorous persuasion he whispered. In five minutes he could phonate, but still stammered, and the next day he spoke normally. There was no relapse and he returned to duty three weeks later.

### Symptoms

In addition to being unable to speak, the patient cannot whisper and instead of the isolated paresis of the abductors characteristic of hysterical aphonia, laryngeal examination shows that the vocal cords do not move at all. We have never found pharyngeal anaesthesia either in hysterical mutism or aphonia, the supposed association being due to unconscious suggestion of anaesthesia on the part of the observer. In the severer cases the patient cannot cough, whistle, or make any sound when he laughs. He may be unable to expire with sufficient power to blow out a candle, and in rare cases he cannot put out his tongue, but he is always able to move it in the act of chewing. Most mute patients keep the mouth closed and make no apparent effort to speak. In other cases they move the lips as if speaking but do not make the slightest sound.

Dumbness is often associated with deafness, especially when it follows exposure to blast, but recovery from either may rapidly occur leaving the other condition as the only remaining symptom. The patient is often at first in a very emotional state and in severe cases he may be in a constant state of terror which is prevented from disappearing by frequent nightmares and constantly recurring mental pictures of the horrors he has passed through.

A mute patient is generally able to convey his thoughts without any difficulty by writing, but in the following case drawing took the place of writing.

*Deaf mutism with ability to draw but inability to write.*—Pte. A., aged 22, was admitted on December 27th, 1916. He could neither hear nor speak. When asked questions in writing about himself he made drawings of trenches, and at the same time became very excited. Suddenly pointing to a prostrate figure in the drawing, he shouted "Bunchy Turner" which proved to be the name of a friend who was killed by his side. With vigorous encouragement he was induced

## CHAPTER VIII

### DISORDERS OF SPEECH

#### HYSTERICAL MUTISM

##### **Ætiology.**

To have one's "breath taken away by surprise" and "to be speechless with rage" express the familiar effects of emotions on respiration and on speech. It is therefore not surprising that loss of speech is a comparatively common symptom in constitutionally nervous soldiers, who have become increasingly suggestible as a result of the stress and strain of active service. Beginning as nothing more than the momentary physical expression of the emotion of fear, when it persists after the disappearance of the exciting cause it is always hysterical. It is generally associated with other hysterical symptoms, such as tremor and less frequently paraplegia, which are also primarily emotional manifestations but may be perpetuated as hysterical symptoms.

Dumbness may be the direct sequel of a single particularly horrible event, as in the following case.

*Hysterical mutism following horror*—A soldier, aged 17, was sleeping in a hut which was blown to pieces by the explosion of a shell. The noise woke him up, and he found that three of his companions had been killed and five wounded. He did not lose consciousness, but ran to a dug-out "speechless with horror." He remained dumb for a week, when speech was restored by suggestion under hypnosis. He continued to stammer for another week, but quickly recovered with re-education and was well enough to return to duty a month after the explosion.

In other cases mutism follows a shell explosion, but it is always emotional in origin and not a result of the concussion. Sometimes the idea of mutism dates from a few moments preceding the explosion.

*Hysterical mutism following blast*—A dumb private admitted under my care on March 3rd, 1917, wrote the following account of himself: "One day, early last October, I heard a shell coming and I tried to dodge, I opened my mouth to shout, but could not, and I then seemed to be blown up. I remembered no more till I was being carried on a stretcher when I tried to shout and could not, and I could not hear. I heard again on the train. I was in several hospitals and

sensation of tension and dryness in the pharynx, a short but frequent hacking cough, and huskiness of the voice. Within a few hours the voice can be produced only with difficulty and finally only in a whisper.

The aphonia at this stage is partly due to a protective reflex, and partly voluntary in order to avoid the pain produced in speaking. If the irritation is severe enough, the patient may decide not to attempt to use his voice at all. This is the best possible thing to do as it gives the inflamed cords a complete rest. Under treatment by vocal rest, with the addition in severe cases of inhalations, the inflammation quickly subsides, and there should be no loss of voice or other throat symptoms after a period of four weeks at most. Frequently however aphonia or even mutism may persist after gassing for much longer periods, even for several months. Our experience showed that all cases of aphonia following gas poisoning which persisted longer than three or four weeks were hysterical in nature and could therefore be speedily cured by psychotherapy whatever the laryngoscopic appearance might be. In a series of 33 cases of gassing without hysterical manifestations, collected by Dr Sylvia Payne, 25 showed throat symptoms on the first day 5 on the second day 1 on the third day 1 on the fifth day and 1 had no throat symptoms at all. In 9 of these cases the throat symptoms lasted a week, 5 two weeks, 16 three weeks, and only 2 for rather longer.

As the result of the stress and strain of war the soldier is especially liable to develop the idea that some permanent damage has been caused to his voice. This idea becomes all the more readily fixed if he believes that his throat is 'delicate' by reason of previous attacks of laryngitis, diphtheria, or tonsillitis. If no improvement occurs in the first few days the patient becomes increasingly despondent and convinced that some permanent damage has been done. When much attention is paid to the condition of his throat at the base hospital and frequent laryngoscopic examinations are made, the fear of incurability becomes more pronounced, and an entry on his medical card of laryngitis, and especially that the vocal cords are congested or otherwise abnormal completes the story of hetero-suggestion.

If, on the other hand, the condition is recognised as hysterical, a rapid and complete cure can be obtained by psychotherapy. A patient, whose aphonia had persisted for two years and eight

any delay, so as to add the more powerful suggestion caused by the pain and contraction of the muscles of the throat following electrical stimulation. When electricity had already been tried elsewhere, but had failed owing to being unaccompanied by a sufficiently vigorous suggestion, a repetition of the treatment with vigorous suggestion sometimes met with immediate success. When it failed I gave the patient ether rapidly. He became very excited and generally talked spontaneously, but an electrode was introduced into the larynx if there was any delay. He was then made to talk continuously until he had completely recovered from the anæsthetic. In two cases the recovery from deaf mutism which followed this treatment was accompanied by partial amnesia.

In the last two years of the war we gave up all forms of suggestion in the treatment of hysterical symptoms, including mutism, as we found that recovery occurred with equal rapidity and with greater certainty after simple explanation followed by persuasion and re-education.

#### HYSTERICAL APHONIA

##### **Ætiology and Pathogenesis.**

Aphonia was one of the commonest hysterical symptoms seen in soldiers in the last war. It may result from any condition which irritates or strains the larynx. Exposure to irritant gases is the commonest cause, but it also frequently follows other inflammatory conditions, such as catarrhal laryngitis, occurring either alone or with pharyngitis or bronchitis, and diphtheria. Actual wounds in the region of the larynx without any damage to the vocal cords are frequently followed by hysterical aphonia.

Aphonia may follow severe emotional strain without any local irritation, but in such cases it is generally a sequel of mutism. In the course of recovery from the latter, whether spontaneous or as a result of treatment, aphonia often occurs, but if properly treated it should not persist for more than a few minutes.

As the majority of the cases occurring in soldiers resulted from exposure to irritant gases, it will be sufficient to describe the mechanism in these cases. Exposure to irritant gases causes an intense inflammation of the pharynx, larynx and trachea, and in some cases of the larger bronchi. Laryngoscopy reveals the presence of all the signs of acute laryngitis—swelling and congestion with muco-purulent secretion. The earliest throat symptoms are a

larynx, of which some followed exposure during the winter and others followed gassing, from these abnormal appearances he concluded that the aphonia was not a neurosis. The patients were consequently treated by intra-laryngeal methods instead of by psychotherapy and it is not surprising to find that in common with most of the other speakers his results were unsatisfactory. If no laryngoscopic examination had been made and nothing abnormal discovered, the cases would have been diagnosed as hysterical and an immediate cure would have followed psychotherapy. I do not mean that a laryngoscopic examination should never be made, but it is necessary to realize that slight abnormal changes in the appearance of the larynx do not preclude the possibility that the aphonia is hysterical. On one occasion I visited the laryngological section of a military hospital and in half an hour cured eight aphonic patients, who had been under treatment for long periods. On another occasion nine cases were transferred to us by a laryngologist, who had had them under his care between six weeks and six months, and they were all cured within 24 hours of admission to the Seale Hayne Hospital. The failure of the laryngologists was due to the doubt inspired in their minds by their laryngoscopic examination as to whether the condition was functional or organic. The success of the psychotherapists was due to the fact that they had made no laryngological examination and were troubled with no such doubts.

### Prophylaxis

It is apparent from the large number of cases of long-standing aphonia seen in England during the last war that the hysterical nature of the complaint was little recognised. A hysterical symptom being one that is curable by psychotherapy no case should persist for more than a few hours after admission to a hospital in England. But aphonia is not only one of the easiest hysterical symptoms to cure it is also one which should never be allowed to develop. If it be recognised that aphonia following gassing and lasting for longer than four weeks is always hysterical a medical officer could induce the patient to speak in a few seconds by simple persuasion if he were still whispering at this period. The same holds good in cases of aphonia due to other causes. Should the aphonia not clear up when the acute stage of laryngeal irritation has passed away the condition should be regarded as hysterical, and should at once be removed by psychotherapy. Of 67 consecutive cases the

months after gassing, was completely cured by Capt. G. McGregor in twenty minutes, although the laryngologist's report was "mucous membrane of larynx much atrophied, vocal cords practically non-existent, this condition is permanent"

The aphonia may be due to flaccid or spastic hysterical paralysis of the laryngeal muscles. In the flaccid variety the muscles, though still capable of contracting, as shown by the fact that they do so if painted by an irritant, remain in the cadaveric position when the patient tries to speak, no trace of adduction occurring. In the spastic variety irregular violent action of the laryngeal muscles takes place, and the inco-ordinated effort may involve all the muscles of the neck and chest. The latter becomes fixed and the patient's face congested and the veins of his neck swollen. Such extreme spasm of the adductor and constrictor muscles may occur on attempting to phonate that both the true and false cords are tightly pressed together, or only the anterior two-thirds of the false cords may meet, and in the gap between their posterior thirds a small portion of the true cords, slightly separated, is seen.

### Diagnosis.

In a discussion at the Laryngological Section of the Royal Society of Medicine in 1918 the President condemned "any arrangement whereby patients suffering from neuroses of the larynx are placed under a medical man who carried out treatment without making a laryngological examination." Believing that many aphonic soldiers are "not suffering from neuroses, but from the result of laryngitis caused by cold or gas, or from weakness, exhaustion, early tuberculosis or paralysis, or they may be simulating," he regarded a laryngoscopic examination as the only means of making an accurate diagnosis. But every one of a series of 67 consecutive cases of soldiers suffering from aphonia proved to be hysterical, whatever the primary cause, as an immediate and permanent cure resulted from psychotherapy (Hurst and Gill, 1919). Of these 37 followed gassing, 18 emotional strain, 8 catarrhal laryngitis, 2 wounds of the neck, and 1 a wound of the chest. We saw no malingerers. Tuberculosis and organic paralysis as causes of aphonia are very rare in soldiers and can generally be suspected from the presence of organic disease elsewhere.

On the other hand, too much reliance on slight abnormalities revealed by laryngoscopy may be harmful. The laryngologist already quoted described various abnormal appearances of the

it is pointed out to him that the noise made in coughing is actually the same as in speaking, so that if a man can cough he can also say "one." He is told to cough again and say "one" immediately after. He often succeeds at the first attempt, and with very little additional encouragement he is soon able to count and talk in his natural voice. It occasionally happens that the voice is at first either hoarse or falsetto. As this is due entirely to inco-ordination of the cords, treatment by re-education should be continued until the natural voice returns, generally within a few minutes.

#### HYSTERICAL STAMMERING

Stammering is a comparatively common war neurosis. It generally follows a severe emotion with or without exposure to a shell explosion. In 1942 I saw a sergeant air-gunner who developed a severe stammer with no other symptom after a crash in which all the other members of the crew were killed. More commonly it is associated with other hysterical symptoms. It is sometimes the primary condition, but more frequently it is a sequel of mutism. In some cases it persists when other symptoms disappear in which case it often does not prevent a man from returning to some form of duty with which his speech defect does not interfere. It is, however commonly associated with anxiety symptoms even in the absence of other hysterical manifestations.

Stammering is also a common sequel of hysterical aphonia, whether this disappears spontaneously or as a result of treatment. If properly treated it only persists for a few minutes otherwise it may be indefinitely prolonged. This form is not associated with anxiety symptoms.

Stammering is particularly likely to develop in men who had some impairment of speech in childhood.

#### Symptoms

Many different forms of stammering occur in soldiers. There may be difficulty only in commencing speech as soon as the first word has been pronounced the rest of the sentence follows easily. More frequently only a few words follow fluently and the patient has to wait again before he can produce a further instalment. One man took two or three minutes to begin a sentence but if, as a result of a great effort, he could say "one" he could then count quickly and intersperse answers to questions without a pause in his counting.

average period under treatment before admission to Seale Hayne Hospital was 205 days, the maximum time being 19 months, the minimum 2 weeks. A pensioner not included in this series had been aphonic for 35 months following tonsillitis, he was cured at a single sitting.

### Treatment.

At the discussion before the Laryngological Society two speakers expressed themselves as opposed to the collection of many cases in one hospital, as in their experience they reacted badly on each other by talking together in whispers. But in a well-conducted hospital they can react only favourably on each other, as no patient is allowed to remain uncured for more than twenty-four hours after admission, so that there are always some who have recently been cured of long-standing aphonia, who encourage the newcomers to believe that they too will be cured at once. For this reason we found that we could be more certain of procuring a rapid result at Seale Hayne Hospital with its atmosphere of cure than when we undertook the treatment of patients elsewhere, although we often succeeded in quickly curing aphonia in other hospitals when the transfer of a patient was inconvenient. Of 67 cases 37 (or 55 per cent) were cured within five minutes, 21 (or 31 per cent) within half an hour, and 9 (or 13 per cent) required more than half an hour.

With increasing experience our methods became very simple. At first we often used electricity as a means of suggestion, applying a faradic current either over the thyroid cartilage or with the aid of an intralaryngeal electrode to the vocal cords, and occasionally we found that the introduction of the intralaryngeal catheter alone without electricity would effect a cure. When these methods failed, as occasionally happened, ether administered rapidly to the stage of intoxication with vigorous suggestion, continued as the patient was coming round until he was wide awake, was generally successful. But during the last year of the war it was only in very exceptional cases that such means were required.

The patient is told that his loss of voice is due to the fact that he has forgotten how to use it. It is explained to him that he whispered after being gassed in order to save himself from pain, as his vocal cords were inflamed, as the inflammation and the pain have now passed away, there is no further need to save his voice. He is then told to cough, as there is rarely any alteration in the cough. His attention is drawn to his natural cough, and

ing of soldiers could be successfully treated by the simple form of psychotherapy which we used for other hysterical symptoms. The following description of the treatment is quoted from a paper by Major J F Venables in the *Seale Hayne Neurological Studies*.

An explanation is given to the patient in language suited to his intelligence of the true mechanism of his stammer and of the circumstances which were responsible for the origin of his symptoms. As muscular spasm is an outstanding feature in all cases of stammering, it is first necessary to obtain relaxation. Then whilst lying at rest, he is taken through the following stages. Quiet respiration is the first stage, at first this may be difficult owing to a kind of respiratory tic, but no attempt must be made to enter the next stage until satisfactory breathing has been established. Excessive depth of inspiration must be avoided, as this tends to produce spasm at the height of inspiration. Quiet phonation during expiration follows the patient is told to repeat first a single word and next short sentences during expiration. The stammerer having once commenced a sentence tends to continue without a break at all costs, as he doubts his ability to begin again. Consequently he attempts to continue speaking when the available air is exhausted or to speak during inspiration. This must be explained, the patient being instructed that he must wait and inspire again the moment he feels any difficulty due to deficiency of air. After he has repeated short sentences, he may practice reading and finally ordinary conversation. It is important to correct certain associated habits, such as word substitution or the use of some short word to introduce all sentences, such as "oh" or "er er". The whole process of re-education may require from half an hour to three or four hours, and in certain cases it is necessary to repeat the treatment for several consecutive days, but in no case should the hysterical stammerer need treatment over long periods. It is necessary to add that the method can be practised only by those who have sufficient patience to continue treatment, if it proves necessary for as long as four hours at a single sitting, and who also have complete confidence in their ability to effect a cure by such treatment.

The following are illustrative cases.

(1) *Stammer dating from childhood associated with aphonia, cured by psychotherapy*—Pte. S., was admitted suffering from aphonia of seven

In other cases speech is extremely slow, deliberate, and drawling, without any actual stammer. An officer with this condition was completely cured after being sent to a quiet spot where he recited and made speeches in the absence of any audience. Very often a syllable or a vowel sound in every word is repeated several times. In other cases the stammerer occupies the time during which he is attempting to begin a sentence by repeating "er er er," as in the speech of a nervous man at his first public meeting. Stammering is often associated with spasmodic movements of the face whenever an attempt is made to speak, in one case these spasmodic movements after a time occurred independently of speech and persisted for a few days after the stammering was cured. Rapid rhythmical side-to-side movements of the head and shrugging movements of the shoulders also frequently accompany the effort to speak.

### Treatment.

If stammering is treated the moment it develops in the course of recovery from mutism, it can always be quickly cured. In my early cases immediate recovery often followed suggestion with the aid of electricity, etherisation, or under hypnosis.

*Stammering cured by suggestion under hypnosis*—Driver D, age 31, was run over by a loaded transport waggon at Gallipoli in May 1915. There was no external injury, but his pelvis was fractured. For three days he was unable to speak at all, although he was perfectly conscious. He then slowly improved, but when he was admitted into the New Zealand Hospital at Walton at the beginning of August, he still spoke with extreme difficulty, and the effort was accompanied by contortions of the whole of his face. Similar contortions of his face also occurred apart from any attempt to speak. They were accompanied by the mental condition characteristic of tic, the patient was able to control them by an effort of will, but he felt miserable when he did so and was always ultimately forced to give way.

He was readily hypnotised, and it was suggested to him that he would be able to speak without difficulty, and that the contortions would cease. The result was very satisfactory, for as soon as he came round he was able to talk quite normally. The next evening he sang at a concert, and a few days later took part in a play.

For a time we gave up direct psychotherapy for the usual methods of teaching respiratory control as used for stammering in civil life. But improvement was very slow and the final result often unsatisfactory. I then recalled the rapid recovery following direct suggestion, which proved that the stammering of soldiers must be a purely hysterical condition, and we found that the stammer-

being stammering. He was employed in England and his stammering gradually disappeared, but he was now unable to play his instrument in the band, the clarinet. With slow music he was as good as before but rapid tonguing was beyond him and had been ever since his return from France. As he was anxious to pass the necessary examinations to become a bandmaster this was of considerable importance to him and he had been practising as much as eight hours a day at "rapid tonguing" but without success. With simple psychotherapy he improved sufficiently in a few days to satisfy himself that he was fit to return to the college and finish his course with a very good hope of passing his examination.

The immediate response of our later cases to simple psychotherapy convinced us that stammering in soldiers was always hysterical, though it was often associated with anxiety symptoms. I believe that many, if not all, cases of stammering in civil life could be rapidly cured by similar means if the treatment were undertaken soon after the first appearance of the stammer.

#### HYSTERICAL HICCUP

Hysterical hiccup is very rare but I saw one case in a soldier which persisted for thirteen months before it was finally cured by psychotherapy (Hurst, 1918).

*Hysterical hiccup of thirteen months duration cured by psychotherapy* — Pte M. was in the trenches on January 7th, 1916 when an aerial torpedo burst four yards away from him. He did not lose consciousness but was much shaken and developed a severe headache. He rested in the dressing station for three days and then returned to duty. On June 19th he sprained his right ankle and was taken into hospital. The ankle remained painful and rigid so that he was unable to walk. He gradually became very nervous, shaky and depressed. On January 20th, 1917 a hiccup developed which continued without intermission except during sleep for thirteen months.

He came under my care in February 1918. He was still continually hiccuping. There were no respiratory movements except those producing the hiccup as respiration consisted entirely of spasmodic contractions of the diaphragm, which I watched with the X rays. When not excited the hiccup was silent and the larynx hardly moved, the rate of hiccup being the normal respiratory rate. But when excited it became very loud, the larynx moved violently and the rate rose to 150 to 200 a minute. He was able to talk only in a spasmodic way, one syllable at a time between the hiccups. Eating and drinking made the hiccup louder and more rapid. He consequently took very little food and became emaciated and weak. The hiccup was aggravated by laughing and by voluntary attempts to control it.

The movements of the left leg were very weak and accompanied by

months' duration following gassing. He was treated the following day in the presence of a class of eight medical officers, and at the end of a few minutes was phonating normally, but stammered badly. He was told at once that now that his voice had returned there was no reason at all for him to stammer, he replied, however, that he had stammered since falling off a bicycle at the age of eleven. Treatment was at once commenced for the stammer, and within half an hour he was talking without any impediment. He remained quite normal for the remaining four weeks he was in hospital, and never exhibited a trace of hesitation.

(2) *Stammer caused by repressed emotional disturbance, cured by the removal of the latter*—Lc-Cpl T had stammered since he was "blown up" in France some months previous to admission. In appearance he differed very much from the first case. Whereas Pte. S was a perfectly normal-looking man, Lc-Cpl T. had all the facial signs of perpetuated fear. Rapid treatment was, however, tried as an experiment, and the result was a temporary removal of the symptom, but stammering recurred the next day when he was paraded with other patients for examination, this had been expected, and a second treatment was given. Ever since he had been blown up he had been unable to go out by himself after dark, as he was liable at any moment to feel a tap on his right shoulder and hear two shrieks behind him. When this occurred he was always so paralysed with fear that his speech left him and subsequently his stammering would be much worse for a time. The same incident frequently appeared in a dream, and he was quite convinced he must be going mad. Further inquiry elicited the following facts. A short time prior to his being blown up his best friend had been killed by his side during an intense bombardment, he had not been killed instantaneously, but had cried out twice and had fallen against Lc-Cpl T's right shoulder. This incident had caused him intense emotional distress at the time, and shortly afterwards he had been himself blown up and lost consciousness. Having recalled this, it at once became possible to prove to him that he was not going mad and to remove his fear of the dark. He now rapidly improved and was discharged with no stammer, no further treatment being required for it, and he had lost his dreams and his "attacks" during the daytime.

(3) *Stammer following spontaneous disappearance of aphonia*—Pte S had developed aphonia with "laryngitis" contracted while dealing with dirty dressings at a base hospital. A sister had gone off duty a few days previously through aphonia supposed to be contracted in the same way. The aphonia persisted for a long time and he subsequently stammered. On admission he had been stammering many months. He was treated as a purely hysterical case and regained his normal speech in the course of forty-eight hours.

(4) *"Clarinet stammer"*—C S M P, a regular soldier, had been in the regimental band before the war, going out with the original B E F. He broke down after many months in France and was returned to England with the diagnosis of "neurasthenia," one of his symptoms

As a result of prolonged re-education he finally learnt to pronounce every word correctly but he still made mistakes, though remaining intelligible if he was inattentive

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clonic spasms, there was also some tremor of the right leg and both arms. A severe degree of talipes equino-varus of the left foot had been present since he sprained his ankle seventeen months before.

He had had a great variety of drugs by mouth and injections for the hiccup but without effect. I tried successively blistering, cold applications and prolonged pressure over the epigastrium but in vain, and inflation of the stomach with air through a tube with the object of fixing the diaphragm in a position of expiration was equally unsuccessful. He was quite unable to carry out breathing exercises, as the hiccup continued without interruption except during sleep, under general anaesthesia and under hypnosis, but it always returned on waking in spite of strong suggestions. Finally he was given sufficient bromide and chloral to keep him semi-comatose for several days. At first the hiccup returned whenever he began to wake, but finally persuasion and re-education succeeded in controlling it when he was confused and half awake. At first it returned when he tried to eat or speak and when he was excited, but at the end of another week it ceased. He was then taught to walk without difficulty, the talipes had already disappeared. He was discharged from the Service in May and went to work on a farm, where he was reported to be getting on well in July.

#### HYSTERICAL IDIOGLOSSIA

Idioglossia was first described by Hale-White and Golding-Bird in 1891, and a thorough study of it was made by Guthrie (1907). It is a condition in which a child speaks a language of his own, substituting sounds for the consonants, but pronouncing the vowels correctly. In severe cases the language is quite unintelligible to everybody except perhaps to the little brothers and sisters of the patient. It is not associated with weakness of intellect or any malformation of the organs of speech. The child generally learns later to speak quite normally. The following case, described by Ripman in 1919, appears to be the only one on record of idioglossia occurring at a later age in a person who had previously spoken normally.

*Hysterical idioglossia in a soldier*—Pt W, aged 42, was blown up in a mine in December, 1916. He was unconscious for seven days and on regaining consciousness was mute. He gradually learnt to speak, but his speech was quite unintelligible. He was admitted to Seale Hayne Hospital two years later. He was depressed, quarrelsome and stupid. His speech sounded like some unknown language, but Ripman succeeded in analysing it. The following are examples of his pronunciation—

pinch—pinks  
god—dod  
glory—jory  
stomach—tomat

glad—jag  
game—dame  
jug—brug  
biscuit—bickit

the war both in gunners and other people who were exposed to the frequent repetition of loud noises, might in the same way become perpetuated and exaggerated by auto-suggestion.

Lastly, organic deafness, especially if the onset is acute, as in that due to involvement of the auditory nerve trunk in cerebro-spinal meningitis, may remain complete after the disappearance of the active disease has been followed by sufficient restoration of the damaged structures for a certain amount of hearing to have returned. Thus, again, is due to auto-suggestion, the final deafness being organic with a superadded hysterical element, which is capable of removal, like all hysterical symptoms, by psychotherapy.

If the deafness remains untreated without any encouragement and especially if steps are taken to teach the patient lip-reading he becomes more and more convinced that he is permanently deaf the effect of the original auto-suggestion being increased by the unconscious hetero-suggestion of those in charge of the patient. One of the worst cases I have seen was that of a man who was told by an aurist that his case was hopeless because he had already been deaf for four months without any improvement occurring. Prolonged psychotherapy was required to cure him. If he had been told that the kind of deafness from which he suffered never lasted for more than four months, and that he would certainly be well in a week, rapid recovery would have resulted.

#### Pathogenesis

Hearing does not consist merely in the perception of impulses conveyed to the brain when the ear is stimulated by sound waves (Hurst, 1920). It is an active process and in order that sounds may be heard the individual must listen. Inattention during a dull sermon results not only in absence of perception of the words said but of total deafness to the sound of the preacher's voice. Keats wrote to a friend that one day during a lecture at Guy's, "There came a sunbeam into the room, and with it a whole troop of creatures floating in the ray and I was off with them to Oberon and Fairyland." Lord Chatfield, in describing his experiences in the *Lion* at the battle of Jutland, tells how his mind became attuned to the salvos of the eight 13.5-inch guns so completely that when he asked his flag-lieutenant why they were not firing more quickly he was told that a salvo had been fired five seconds before he had spoken although he had not heard it. It is clear that impulses to the cortical centre of hearing must be actually

## CHAPTER IX

### FUNCTIONAL DISORDERS OF HEARING

#### HYSTERICAL DEAFNESS

*Falstaff* it is a kind of deafness

*Chief Justice* I think you are fallen into the disease, for you hear not what I say to you

*Falstaff* Very well, my Lord, very well, rather an't please you, it is the disease of not listening, the malady of not marking, that I am troubled withal.—*Henry IV*, Part II, Act 1, Scene 2

The noise and concussion produced by the explosion of a shell of high power in the near neighbourhood frequently caused deafness. The patient was dazed or unconscious as a result of the explosion, and when his mind became clear again he discovered that he could not hear. Both ears were generally affected, but the one on the side more exposed to the explosion of the shell was often deaf than the other. The initial deafness was doubtless due to concussion of the internal ear, as it was sometimes associated with vertigo and temporary nystagmus. One or both drums were often perforated owing to the sudden enormous change in atmospheric pressure. Such a perforation did not greatly affect the prognosis, as the tear generally healed, and if no hysteria developed normal hearing was restored. Sometimes, perhaps, the ossicles were dislocated by the force of the explosion, in which case some permanent impairment of hearing would result.

The deafness might pass off in the course of a few hours, but more frequently it lasted for a few days. If it persisted for a still longer period, it was almost always hysterical, at any rate in part. The initial concussion deafness made such an impression on the mind of the soldier that, on coming to himself, whether he had actually lost consciousness or not, his first thought was for his hearing, and he might be so convinced that he was permanently deafened that he became actually deaf as a result of auto-suggestion. This was especially likely to be the case if for any reason the idea of deafness had previously entered his mind, it was for this reason that a large proportion of cases occurred in men who had old disease of the ear.

The temporary deafness, which was a familiar condition before

noise which induces it even in a dream. The afferent part of the reflex is thus also sub-cortical the reflex being quite independent of actual hearing.

Experiments on animals by Sherrington and Forbes (1914) confirm the conclusion we reached from clinical observations—that the auditory motor reflex is a function of the mid brain. They showed that both the posterior corpus quadrigeminum and the medial corpus geniculatum are concerned. The close relation of these centres to the blinking and the sympathetic pupilo-dilator centres in the neighbourhood of the third nerve nucleus, and to the anterior corpus quadrigeminum and red nucleus, in each of which a descending motor tract originates, gives an anatomical basis for this view. In the experiments of Sherrington and Forbes on cats, sounds, especially barks, yowls, and whistles of birds, excited orientatio reflex movements of the pinna and neck after the complete removal of the cerebral hemispheres, striata, and thalamus. Reflex movements expressive of anger and aggression—lashing of the tail with bristling of its hairs, and flexion and extension movements of the limbs—were also produced. In our cases of complete bilateral deafness the auditory motor reflex was completely absent, but returned simultaneously with the restoration of hearing as a result of psychotherapy. A very nervous but totally deaf mute remained completely unmoved never jumping or showing a flicker of his eyelids during one of the severest thunderstorms I have known, and yet the next day he was completely cured by suggestion under partial anaesthesia.

A slight reflex was present in most cases of severe but incomplete hysterical deafness, but it became less marked and sometimes disappeared completely or was confined to a slight dilation of the pupil when the test was repeated.

The abolition of the auditory motor reflex in absolute hysterical deafness makes it clear that one or more of the unswitched synapses in hysterical deafness must be at the level of the reflex, or still lower—in the auditory nucleus or one of the intermediate cell-stations, the superior olive and the nucleus of the lateral fillet, or perhaps in all.

The persistence of the deafness during hypnosis and natural sleep shows that when the inattention of hysterical deafness has lasted for a considerable period, the unswitching of the synapses is more profound than that which normally occurs during deep sleep in which the synapses can always be forced by a loud noise.

interrupted by inattention. The most satisfactory theory is that in the act of listening the resistance at the various synapses in the auditory path becomes diminished by some such process as a throwing out of dendrites, which brings those of contiguous neurones into more intimate connection. In inattention the synapses are unswitched, the resistance being increased by the retraction of the dendrites.

When a man is temporarily deafened by a loud noise or by some recoverable disease, he finds that he is unable to hear, however much he tries, and consequently after a time he gives up trying. That is to say he ceases to listen, and when the cause of the deafness at last disappears, he has become so convinced that he cannot hear that he makes no further attempt to listen. Although the sound vibrations reach his ears in the normal way, they do not give rise to the slightest auditory sensation because of this inattention: he is then suffering from hysterical deafness, the inability to hear having been suggested by the original organic, though temporary, deafness. Severe hysterical deafness developed in a soldier while he was the only Englishman in a German prison, he ceased to pay attention to what was said, as he could understand nothing, and in time he ceased even to hear the unintelligible conversation of his companions. His hysterical deafness was rapidly cured by psychotherapy when a year later he came into Seale Hayne Hospital.

In hysterical deafness the synapses at one or more of the cell-stations in the auditory path to the cerebral cortex must therefore be unswitched, possibly as a result of retraction of the dendrites. Further evidence for this follows from a study of the auditory-motor reflex.

*Auditory-motor or jump-reflex*—A sudden noise normally causes an individual to jump and often to blink, and the pupils dilate, the "jump," at any rate, is a protective reflex, and represents the preparation for flight or fight.

An officer, whose left motor cortex had been almost completely destroyed, went to the *Man who Stayed at Home* about four months after he was wounded. His right arm jumped violently when the gunshot rang out on the stage, although no trace of voluntary movement returned until three months later. The efferent part of the reflex is thus sub-cortical. In certain war neuroses of emotional origin, in which the reflex is exaggerated, jumping continues during sleep and deep hypnosis, although the patient does not hear the

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frequently in otherwise normal individuals with no symptoms or history of neuroses.

(3) *The supposed association of hysterical anaesthesia of the external ear with hysterical deafness*—Ever since Briquet in 1859 described the association of hysterical deafness with anaesthesia of the external ear this has been regarded as an almost constant phenomenon.

Symms Gainsborough, and I (1918) noted the sensibility of the external ear in a series of 29 soldiers suffering from organic deafness of varying origin and of varying degrees of severity. The ears were first touched lightly with a finger or some wool sometimes beginning with the deafer ear sometimes with the ear which was less deaf or normal, at the same time the patient was asked, "Which side do you feel more distinctly?" A similar comparison was then made between each ear and the cheek on the same side. In 28 out of the 29 cases hearing was distinctly better on one side than the other and in several the latter was normal. Eighteen of the 28 had partial anaesthesia, and 4 had hyperaesthesia of the affected ear—the better ear was normal in every case. In the single case in which the deafness was equal in the two ears there was no anaesthesia. But this man did not appreciate that he was deaf at all, a slight degree of deafness having been discovered only when the ears were examined on account of other symptoms. The 6 cases of asymmetrical deafness, in which no anaesthesia or hyperaesthesia was found at the first examination, were then examined again, but a gross form of suggestion was now employed, the patient being asked the following questions: "You feel my finger less clearly when I touch this ear (the deafer one) than this one, don't you?" and "You feel me touch this ear (the deafer one) less clearly than your cheek, don't you?" Four out of the six patients answered

Yes—in the remaining two the degree of deafness was very slight. In several of the patients anaesthesia was found to be very well marked and was associated with complete or partial analgesia. So real was this loss of sensation that one patient was subsequently seen putting a pin through the lobe of his ear for the amusement of the other men in his ward.

In a case of severe bilateral hysterical deafness examined in the same way both ears were found to be completely anaesthetic. In a second case, in which one ear was totally deaf owing to organic disease and the other had been totally deaf owing to hysteria, but in which partial recovery had occurred as a result of psychotherapy

### Diagnosis.

In the observations I carried out at Netley with E A Peters (1917) we found it necessary to discard almost all the criteria formerly used in the diagnosis of organic deafness from deafness due to hysteria or malingering

(1) *History*—Complete bilateral deafness following the explosion of a powerful shell was generally hysterical, though a lesser degree of asymmetrical organic deafness of a more or less permanent nature might be produced at the same time owing to perforation of the drum or hæmorrhage into the middle ear, both of which were often followed by otitis media, or to dislocation of the ossicles. It is doubtful whether absolute organic deafness ever results from hæmorrhage into the internal ear following aerial concussion, as no anatomical evidence of such an occurrence was ever forthcoming. Deafness following an acute illness, especially cerebro-spinal meningitis, is at any rate in part always organic, but we have found that the deafness may be increased as a result of auto-suggestion, no spontaneous improvement in function occurring when the anatomical condition improves

(2) *Other evidence of hysteria*—Our investigations have shown that hysterical symptoms may develop in the most normal individuals if the suggestion which provokes the symptoms is sufficiently powerful. No more powerful suggestion of deafness could be imagined than the organic but temporary deafness caused by the noise of an explosion in the immediate neighbourhood. It is therefore not surprising that hysterical deafness has occurred almost as frequently in soldiers who have no personal or family history of neuroses as in those with a neuropathic predisposition. Deafness has consequently been the only hysterical symptom in many of the cases. When, however, the onset was associated with extremely terrifying circumstances, some of the physical results of fear, such as mutism and tremor, were often perpetuated as hysterical symptoms. Thus hysterical deaf mutism was quite common. In two cases sand was thrown into the patient's eyes from the sand-bags on which the shell exploded, and the hysterical deafness was accompanied by hysterical blindness, which followed the conjunctivitis caused by the irritation of the eyes with the sand.

The only conclusion which can be drawn from these facts is that while the association of deafness with mutism or with tremor is very suggestive of hysteria, hysterical deafness occurs most

heard at all, it may produce a reflex. If a reflex is present, but the patient says he can hear nothing at all, he is probably a malingerer but so long as he admits that he can hear something the test does not help in distinguishing between organic deafness, hysterical deafness, and malingering.

(6) *Persistence during sleep*—As hysterical symptoms are the result of suggestion, it might be expected that they would not persist during sleep and Babinaki regarded this as a definite law. My experience agrees with his with regard to all other hysterical symptoms which I have investigated, such as paralysis, contractures and anaesthesia. I have seen several deaf mutes and one aphonic patient who talked naturally in their sleep and a man with hysterical amnesia who had nightmares referring to his period of amnesia. But hysterical deafness is an exception. Thus I found it quite impossible to wake two of my patients, who were suffering from total hysterical deafness and were sleeping in a hut by themselves by shouting or by making other very loud noises within a foot of their heads. I convinced myself that deception was impossible and the hysterical nature of the deafness in both cases was at a later date conclusively proved by their instantaneous recovery with powerful suggestion. In one patient a slight twitch of the eyelids was sometimes observed with a particularly loud noise but not in the other. It seems probable that a malingerer could be detected by this test, as he would certainly wake if a loud noise were made when he was asleep.

(7) *Effect of hypnosis*—I had expected that hearing would return in hysterical cases during hypnosis, but I found it quite impossible to make deaf patients, whom I had deeply hypnotised, obey any command or show any signs of hearing, and no auditory-motor reflex was produced. The unswitched synapses thus appear to remain unswitched during hypnosis, as they do during sleep.

(8) *Character of the voice*.—In almost all cases of severe deafness due to organic disease the character of the voice changes. It is difficult to understand why there should be any difference in the effect of total deafness on the voice, whether it is organic or hysterical as the change is simply a result of the patient's inability to hear his own voice. Although in some of our hysterical cases the typical voice of the organically deaf developed, the majority showed no change in timbre or intonation.

(9) *Lip-reading*—When a deaf man teaches himself lip-reading,

the organically deaf ear was found to be anæsthetic, and the hysterically deaf ear was normal, corresponding with the fact that the deafness of the former was much more severe than that of the latter

It is thus clear that the supposed association of hysterical anæsthesia of the external ear with hysterical deafness is a complete fallacy, and that anæsthesia is likely to occur in a deaf ear if looked for, whether the deafness is organic or hysterical, so long as the individual is sufficiently suggestible and not too well educated. In all the above cases the patients were soldiers who had been on active service, and our experience showed how suggestible the majority of war-worn soldiers are, even in the absence of any hysterical or other nervous symptoms. The results obtained with ordinary hospital patients were consequently less striking. Among four adult male civilians, three females, and two boys, all of whom were deaf in one ear or deeper in one than in the other, only one of the adult males had an anæsthetic external ear.

Ten well-educated individuals, who were asked whether they would expect any difference between the sensibility to touch in the two external ears if they were deaf on one side only, replied in the negative. On the other hand, seven indifferently educated men all replied in the affirmative. The difference is simply due to the fact that no intelligent man would expect his external ear to be anæsthetic if he were deaf, but a man of less education would act upon the suggestion implied in the question without criticising it. The greater frequency of grotesque hysterical symptoms among hospital than private patients, and among private soldiers than officers, is doubtless due to the greater intelligence and greater development of the critical faculties to the latter.

(4) *Bone and air conduction* —When deafness is not absolute, a tuning-fork can still be heard by bone conduction (positive Rinne's test). This shows that the deafness does not depend on changes in the middle ear, even when these are present. But it does not distinguish between the nerve deafness due to organic disease and that due to hysteria. Moreover, the test can be applied only in the slighter cases, as in many instances deafness is absolute and no hearing is possible, whether the sound is conducted by bone or through the air.

(5) *Auditory-motor reflex* —In absolute deafness, whether due to organic disease or hysteria, the auditory-motor reflex is absent, but in partial deafness, whether organic or hysterical, if a sound can be

some sixteen days now since it happened. We were in the trenches and going for dear life when two of us spotted a German machine gunner in a hole so we made up our minds to have him. We made a charge at him and I just remember getting to him when a high-explosive shell burst at my head—it seemed as if it burst inside my head—everything went black. I tried to call out and couldn't, and I could not hear my mates—only just a terrible bursting in my head all the time. I never remembered anything more until I came to on the boat. The doctors have told me that I will get all right in time.

The letter ended abruptly at this point, as I then came to examine him. The previous day I had hypnotised him without difficulty but was unable to make any effective suggestions, as the deafness persisted during the hypnotic sleep so that the suggestions did not reach the higher centres of his brain and were consequently not acted upon either whilst he remained asleep or after he awoke. He was so deaf that he heard nothing at all during an exceptionally violent thunderstorm. He was not only unable to speak, but could make no sound of any kind and could not cough.

As no improvement had taken place, he was given ether after being told in writing that it would have the effect of restoring his speech and hearing. He began to struggle after a first few whiffs and long before he was anaesthetised he began to repeat the word *Mother* first in a whisper then louder and louder until he shouted it with a stentorian voice that would have filled the Albert Hall. The etherisation was then discontinued, his limbs never having become relaxed. As he came round, I told him to say various words which he repeated after me and I then carried on a continuous conversation with him. When the effects of the anaesthetic finally passed away he was talking with a normal voice and he had completely recovered his hearing.

Uncomplicated hysterical deafness is much more difficult to treat, and for a time our results were less satisfactory than in any other hysterical condition. For this reason we were induced to employ various forms of suggestion, although we realised that the methods were not really satisfactory. Suggestion under hypnosis was never of any use, as the patient remained deaf while hypnotised, and consequently heard none of the suggestions which were made to him. Electricity as a means of suggestion was sometimes successful, but often failed. In a number of cases of absolute bilateral deafness, which had resisted all other forms of treatment for some months, we performed "fake" operations, making a scratch behind the ear in the incompletely anaesthetised patient.

*Hysterical deafness following exposure to a shell explosion cured by a fake operation.*—Lance-Corporal M. aged 28 was blown up by a shell on August 29th, 1916. He became completely blind, deaf and dumb although he did not lose consciousness. His sight returned the following

his deafness must be of a high degree. Several patients with hysterical deafness learnt it with remarkable rapidity, so that it is not, as has been suggested, a sign of organic disease. On the other hand, it is very unlikely that a malingerer would ever learn lip-reading.

(10) *Vestibular symptoms and reactions*—Disturbances in the vestibule as a result of concussion may cause spontaneous nystagmus, which may be accompanied by giddiness and staggering, but these symptoms rarely last for more than a few hours.

We found that the only test upon which almost complete reliance can be placed in the diagnosis of absolute hysterical deafness from absolute organic deafness is the presence of normal vestibular reactions to the rotation and caloric tests in the former and their loss in the latter. The vestibular reactions are entirely beyond the control of the will, and it is therefore inconceivable that they should disappear as a result of suggestion. As hysterical symptoms are always caused by suggestion, the vestibular reactions must remain unaffected in hysterical deafness. On the other hand, it is highly improbable that any organic lesion could damage the cochlea or the cochlear nerve or nucleus on both sides sufficiently to cause total bilateral deafness without at the same time damaging the vestibules or vestibular nerves or nuclei, so that total organic deafness is almost certain to be accompanied by deficiency in the vestibular reactions.

### Treatment.

When hysterical deafness is associated with mutism it requires no special treatment, as hearing almost invariably returns spontaneously when speech is restored. In order to make this still more certain, the patient must be convinced that directly he speaks he will hear his own voice, and that he will then hear everything clearly. There is rarely any difficulty in curing the mutism by simple explanation and persuasion, though in our earlier cases we occasionally resorted to the suggestion produced by applying faradism to the larynx or making the patient excited with an anæsthetic.

*Hysterical deaf-mutism following shell explosion cured by suggestion with etherisation and followed by partial amnesia*—An Australian soldier, aged 22, wrote the following letter to his relations on August 21st, 1916: "You may be a little surprised to hear that I am in the hospital suffering from shell-shock, which has taken away my speech and hearing. It is

his deafness had now disappeared as the deafness was at first organic, he could not hear however much he listened and consequently after a time he had ceased to listen at all. He was next persuaded to listen intently and was taught that listening was just as active a process as moving and required a conscious effort on his part until it again became automatic. This method was successful in a case recently seen with Dr Harold Davis.

*Hysterical deafness produced by gunfire oil drops and a cocktail. Recovery three years later with psychotherapy*—The patient a young officer was proud of his exceptional powers of hearing when he joined the Army in 1934. He had found that he had such perfect hearing that he could "hear a watch ticking on a fence in an open field thirty feet away. The first time he fired a shot on a rifle range he was distressed to notice that his hearing in both ears became impaired. He consulted a doctor who gave him some oil as a preliminary to having them syringed. The moment the cold drops of oil entered his left ear it became completely deaf so he injected none into the right. When next day the left ear was syringed no wax came away and there was no improvement in hearing. The patient himself attributed the deafness of the left ear to the shock of the cold oil against the drum. He was advised to resign his commission as he was told that gunfire would be likely to impair his hearing still further. He did not do so but went to France with the Expeditionary Force. The noise during the fighting in Flanders and France caused considerable deterioration, but he could still hear a little with his right ear.

The patient was teetotal because of a promise made to his father that he would neither drink nor smoke till he was twenty-one. One evening at a party soon after his return to England he was persuaded to drink a cocktail. He had swallowed a single sip when the hearing in his right ear suddenly disappeared almost completely and the music from a loud band playing on the wireless changed to a faint noise without any musical tone. He had previously had a very good ear for music but with the very slight hearing he still retained in his right ear he could not recognise any tune or differentiate between two notes loudly played on the piano.

He consulted numerous ear specialists all of whom told him that he had nerve deafness and that nothing could be done for him. He was advised to get an electric hearing-aid. The moment it was switched on his hearing became still worse. He was much annoyed and the attendant sent him in alarm to another aural surgeon who like all the others, told him that no treatment was likely to benefit him. It was never suggested that the deafness might be wholly or in part hysterical until he happened to meet Dr Harold Davis, a general physician who had read about cases of hysterical deafness in the war of 1914-18 and felt convinced that this patient must be suffering from the same condition.

When I first saw him in June 1943 with Dr Davis, he had no hearing

day On reaching England he was able to read and write, and he talked in his sleep In spite of treatment with encouragement, electricity and etherisation, no further improvement occurred until one night in November, when he woke up and asked the Sister for a drink After this he was able to talk normally, but the deafness remained.

He came under my care at Netley on March 21st, 1917, seven months after the onset of the deafness He was found to be completely deaf both to air and bone conduction, though he could feel the vibration of a tuning-fork on his mastoids A loud noise just behind his head caused a slight tremor of his hands, blinking and dilatation of the pupils, although he heard nothing A slighter reaction was produced on the second and third occasion when the noise was repeated After this it disappeared completely and did not return

As the vestibular reactions were found to be unaffected, it seemed probable that the internal ear was free from organic changes This was rendered still more likely by the fact that immediately after the explosion the deafness was associated with mutism The patient was hypnotised, but the deafness persisted he could not be made to respond to any suggestion, as he was unable to hear, and a loud noise produced no auditory-motor reflex, the pupils as well as the eyelids remaining fixed The deafness also persisted during natural sleep, as it was found impossible to wake the patient by shouting "fire" and by banging a poker against a coal-scuttle within a few inches of his head, and no reflex flicker of his eyelids was observed In the morning he had no recollection that anything unusual had occurred during the night He was suddenly and completely cured by a "fake" operation on his ear on April 20th, to his intense delight, as he had recently become extremely depressed at the absence of any sign of improvement after more than seven months His hearing was accurately tested the next day, and it was found that it was perfectly normal both to air and bone conduction, and the auditory-motor reflexes had returned He was discharged to duty three weeks later, feeling perfectly fit He visited the hospital on June 29th, a few days before he returned to France, his hearing was normal, and he was well in every way

This method of treatment was not invariably successful, and at the best it was not one which could be regarded as desirable, as it is much more satisfactory for the patient to understand the exact means by which he has been cured than for him to be fooled into a cure by gross suggestion. With increased understanding of the psychological basis of hysterical deafness we were able during the last year of the war to cure a large majority of cases by the most rational form of psychotherapy—explanation, persuasion and re-education The patient was made to understand by written explanations how he had become deaf, and how the original cause of

have become timid owing to their resisting power having been worn out by prolonged mental strain, their special senses and the reflexes associated with them becoming tuned up to such an extent that sights and sounds, which men with stronger nerves learn to disregard after they have been in the trenches for some time, give rise to more and more exaggerated motor responses.

The patient is abnormally sensitive to sound and hates the slightest noise—he is unable to sleep in a town, and sudden noises often frighten him. The condition of most patients suffering from war neuroses is much aggravated by a thunderstorm or the sound of distant firing, as during an air raid. Many are reduced to tears, and few are able to sleep the following night. Bright light is also disagreeable, but vision is less sensitive than hearing. In most cases these symptoms are simply due to increased irritability of the nervous system, but in one severe case true hyperacusis was present, and Captain E. A. Peters and I estimated that the patient heard sixteen times more acutely than the average normal individual. It was possible to carry on a conversation with him by whispering in one corner of the ward when he was lying in the opposite corner, although men with normal hearing who were standing half way between in the centre of the room could not hear a word of what was whispered. The hyperacusis and exaggerated defence reflexes were quite uninfluenced by the administration of 100 grs. of bromide a day and were only slightly reduced by plugging the ears with plasticine.

Most men rapidly improve when removed from the sights and sounds which caused their abnormal state to develop and the defensive reflexes become less and less exaggerated. If however the emotion of terror is kept alive by vivid dreams, and still more if the individual continues to picture the horrors he has witnessed to himself even when he is awake and every sound reminds him of the bursting of shells, the symptoms persist.

The flinch reflex now occurs whenever anybody even the patient's friends and relations, comes near and the jump reflex, which may at first be produced by nothing less than an explosion in the near neighbourhood, now follows other noises, until in time quite harmless and comparatively faint sounds give rise to it.

In severe cases the patient appears to be in extreme terror. He jumps violently and trembles from head to foot at the slightest sound—he raises his arm as if to protect his face from a blow and

at all in his left ear. With the right ear he could hear sounds very faintly, but he could not distinguish words or notes of music. He was able to carry on a conversation fairly well by means of lip-reading, in which he had become very expert. His story pointed clearly to an hysterical origin. Being specially proud of his hearing he was likely to be unduly impressed by the slight deafness which naturally results from gunfire, so that it did not quickly disappear in the normal way. The sudden aggravation after having cold oil dropped into his external ear could have no organic explanation, and the emotion connected with drinking the forbidden alcohol further aggravated the deafness. We explained the nature of the deafness to him, and gave him lessons in listening to conversation with his eyes covered to prevent lip-reading and also to the piano. He rapidly improved, but was able to stay in Oxford only long enough to have four lessons. He continued to practise listening at home, and by October was hearing sufficiently well with his right ear to be able to hear everything said to him in a voice of ordinary loudness at a distance of ten feet. The listening required for hearing still required a voluntary effort instead of being automatic. He is now training his left ear in a similar manner.

#### EXAGGERATED FLINCH AND JUMP REFLEXES, HYPERACOUSIS

The "flinch reflex," which is caused by the sight of danger approaching, consists in the assumption of a crouching attitude, the arm is raised in front of the face and the eyes blink. It has the object of hiding the individual and defending him from attack. The "jump reflex" consists of a sudden movement of the limbs and trunk, and is associated with blinking of the eyes and dilatation of the pupils, it is caused by sudden sounds, and is really the preparation for the immediate activity, which would be displayed if the individual obeyed his instinctive desire to save himself by flight, the special conditions at the front lead to ducking of the head being added to the ordinary jump reflex of civil life. Our observations and Sherrington's experiments on animals show that these responses are true defensive reflexes and have their centres in the mid-brain.

The common exaggeration of the jump reflex in soldiers suffering from certain war neuroses is not, therefore, correctly described as hyperacousis, for the sense of hearing may be no sharper than normal, and actual hearing need not occur at all, as the reflex occurs when a patient is asleep and hypnotised as well as in some cases of hysterical deafness. It is simply a part of the general exaggeration of the defensive reflexes, which is a characteristic feature of those war neuroses which are purely emotional in origin. It is specially common in men who are constitutionally timid or who

## CHAPTER X

### FUNCTIONAL DISORDERS OF VISION

#### HYSTERICAL BLINDNESS

In Chapter IX it was shown how in order to hear it was necessary to listen, and that the process of listening involved some change at each synapsis in the auditory tract from the cochlea to the cerebral cortex, as a result of which resistance to auditory impulses was diminished.

In order to see it is necessary to look. Looking involves a process in the visual tracts strictly comparable to what occurs in the auditory tracts in listening. But, in addition to this, the action of a number of muscles is called into play. Two distinct elements thus require consideration—the afferent, involving the visual tracts, and the efferent, involving certain motor tracts.

(a) *The Afferent Element*—Inattention is rarely so profound in normal individuals that no visual impulses reach the brain at all, although complete absence of hearing not infrequently results from extreme inattention. It is common enough for visual impulses to be so blurred that a man, wrapped up in his own thoughts, does not see the approach of a friend, but he would always avoid obstacles if he were walking, and he would be aroused by the sudden approach of an object to his eye. The latter would also give rise to the flinch reflex.

In the condition of stupor which was not uncommon in soldiers who had been exposed to exceptionally terrifying ordeals, the mind appeared to be so completely absorbed with thoughts which had no connection with the patient's present surroundings that he did not respond to any impulses from the outside world. He appeared to be blind, deaf, and anaesthetic—he gave no flinch (visual motor) or jump (auditory motor) reflex, cutaneous (tactile-motor) reflexes were often, though not invariably abolished, and the pupil contracted sluggishly or not at all on exposure to light. In spite of this he would feed himself if food were put in front of him, and he did not stumble against obstructions if he were taken for a walk, and he occasionally showed a slight response to certain loud sounds, so that vision and hearing were clearly still possible but it was

hides his head under the bedclothes when anybody approaches him. After all conscious feeling of terror has disappeared and the patient no longer thinks or dreams of the horrors of war, the appearance of terror may continue unabated.

#### TINNITUS

Tinnitus may be associated with hysterical deafness and may continue after the disappearance of the latter, but it also occurs independently. It is frequently noticed by the patient only after some improvement has occurred in the severe deafness immediately following an explosion in the near neighbourhood.

*Tinnitus resulting from blast*—A man admitted for symptoms resulting from the explosion of a high-explosive shell complained when he regained consciousness of constant noises in his head, which never ceased and greatly distressed him. His hearing was normal, but I found a thick hair growing in the external meatus close to the drum on each side, it touched the drum on the side in which the noises were loudest and the opposite wall of the meatus on the other side. The removal of the hair was immediately followed by disappearance of the tinnitus. The hairs must have been present for years, but until the irritability of his nervous system became exaggerated by emotion caused by the explosion they had caused no trouble. An alternative explanation is that the hair was driven in so as to touch the drum by the force of the explosion.

Tinnitus may result in auditory hallucinations. The patient imagines he hears shells coming towards him and bursting, or he may hear the whizz of bullets or the blowing of whistles.

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thorough co-ordination, so that the object to be looked at is brought into the centre of the field of vision of both eyes, and the ciliary muscles contract just sufficiently to bring it into correct focus. All these motor processes may be impaired as a result of suggestion either alone or in addition to the afferent element already described. How this occurs can best be understood by a consideration of the hysterical disorders of vision which follow gassing.

Exposure to mustard gas is followed in a few hours by pain in the eyes, which is increased by exposure to light. Conjunctivitis, blepharitis, and in rare cases keratitis, quickly develop. As long as pain and photophobia are present, the swollen lids remain closed. This is partly due to inactivity of the levator palpebræ superioris muscles, for the patient makes no effort to open his eyes, knowing that if he were to do so the exposure to light would cause pain. It is partly due to a protective reflex, which results in over-action of the orbicularis palpebrarum muscles, particularly if the patient does try to open his eyes. The object of this is to protect the eyes from being irritated by light—it is accompanied by reflex lachrymation as a result of which irritating material is washed away. The inflammation gradually subsides, the reflex blepharospasm and lachrymation disappearing *pari passu*. At the same time the majority of patients realise that they can now open their eyes without hurting them, and in most cases they do so.

In individuals, however, who have become abnormally suggestible as a result of the stress and strain of active service and in others who for any reason are particularly anxious about the condition of their eyes the normal results of the conjunctivitis become perpetuated by auto-suggestion, to which may sometimes be added the hetero-suggestion caused by injudicious treatment, such as the prolonged use of eye-shades and dark glasses, which give rise to an exaggerated fear in the patient's mind of the consequences of having been gassed. The voluntary inactivity of the levator palpebræ superioris becomes perpetuated as an involuntary inactivity which may amount to actual paralysis, the condition being now one of hysterical ptosis. In rare cases the hysterical paralysis of the levator palpebræ may spread, hysterical paralysis of the whole of the face resulting (Fig 15). A patient who finds himself unable to open his eyes by contracting his levator palpebræ muscles often attempts to compensate for this by contracting his frontalis muscles, and if the attempt meets with sufficient success to enable him to see through

difficult or impossible to induce him to look or listen even for a few seconds at a time. Exactly the same thing occurs in somnambulism. The doctor, seeing Lady Macbeth walking in her sleep, exclaims, "You see, her eyes are open," and his companion replies, "Ay, but their sense is shut."

In the chapter on Hysterical Deafness it was explained how the idea of being unable to hear, suggested by temporary organic deafness, might give rise to a continued absence of listening and consequent deafness after the organic cause had disappeared. In the same way any condition, which has led to complete though temporary blindness, may suggest to the individual that he has lost his sight for ever. This is particularly likely to be the case if the temporary blindness is produced suddenly under terrifying conditions, as, for example, by the explosion of a powerful shell in the immediate neighbourhood. The slower onset of the temporary blindness in gassing, although the surrounding conditions might be equally terrifying, generally resulted in less profound hysterical blindness. When the suggestion that the sight is permanently lost has become thoroughly accepted, the individual will cease to look. The visual tract is no longer prepared for sight by attention, and visual impulses consequently cease to pass to the occipital cortex. In the act of looking the resistance at each cell-station in the tract appears to be diminished by some such process as a throwing out of dendrites or an alteration in the electro-chemical condition of the synapses. Consequently visual impulses not only give rise to no visual perception, but the flinch reflex and in the most severe cases even the pupil reflex to light disappear, as the resistance to the impulses, even at the lowest synapses, is too great to be overcome. More commonly the impulses produced by a very bright light can still break through the resistance, so that the light is perceived and a sluggish reflex to light is obtainable, but the flinch reflex is still completely abolished.

When, as a result of psychotherapy, the patient realises that he can really see if he only chooses to look, he once more throws out the dendrites or otherwise reduces the resistance in the visual path, and vision returns, the pupil and flinch reflexes becoming normal again at the same moment.

(b) **The Efferent Element.**—With the afferent element set in readiness, visual impulses reach the occipital lobes, but nothing is seen clearly until the eyes are opened, the extrinsic muscles work in

does not contract them to the correct extent. He has in fact lost control over accommodation, and believing himself blind, looks at nothing.

The external ocular muscles may remain inactive with the production of hysterical external ophthalmoplegia—a very rare condition, which I have however seen occasionally. Much more frequently certain muscles are thrown into spasm in an attempt to bring them into action after long disuse and hysterical strabismus results. Thus hysterical spasm of convergence is often seen when the eyelids are forced open, in addition to the contraction of the superior recti, which tends to keep the pupils hidden under the upper lids. In consequence of these abnormalities of accommodation and convergence, the patient sees nothing clearly, but he is not completely blind. Complete hysterical blindness which is rare in cases of this kind, is due to the patient being so convinced he cannot see that he does not use his visual centres at all. The mechanism of this condition has already been described.

The following cases of hysterical disorders of vision have been selected from the large number I have seen as illustrating what has already been said concerning their pathogenesis.

The first case is of exceptional interest as it is also the only one in which the reflex contraction of the pupils to light was abolished. So far as I am aware no similar case had hitherto been described.\*

*Total blindness with loss of pupil reflexes following shell-concussion hysterical nature proved by rapid cure by psychotherapy after persisting for four years*—Pioneer B. aged 41 went to France in September 1914. After six weeks fighting he was stunned as the result of the explosion of a shell in his immediate vicinity. In the evening he noticed he could not see clearly and attributed this to the shock of the explosion. He at once feared he would lose his sight—his eyelids began to droop and he had difficulty in focussing near objects. He was sent to England, where the use of eye-drops and dark glasses confirmed his fears, and in a short time he became totally blind. Early in 1915 he was discharged as permanently unfit, receiving a full pension for total blindness. He was examined every six months after this, but no treatment was given. In November 1918 he was seen by Mr J. R. Rolston of Plymouth, who recognised the condition as hysterical, and advised his transfer to Seale Hayne Hospital, where he was admitted on November 15th, 1918. He presented the picture of the typical blind beggar

\* An exactly similar civilian case was under my care in 1930 and was cured by Dr D. Ellis by simple psychotherapy (*Gay's Hosp. Rep.*, 50 26.)

the chinks between his eyelids, he does this more or less constantly. The reflex blepharospasm is perpetuated as hysterical blepharospasm, which is generally most obvious when the patient attempts to open his eyes voluntarily, the eyes becoming more tightly closed than ever. The paralysis of the levator is thus accompanied by spasm of the orbicularis, just as hysterical paralysis of one group of muscles in a limb may be accompanied by hysterical spasm of the opposing group of muscles. The spasm often involves the neighbouring muscles or even all the muscles supplied by the facial nerve. Any attempt to pull the eyes open meets with great resistance. In some cases, after it has become possible to open the eyes voluntarily, the ptosis and blepharospasm manifest themselves intermittently in the form of attacks of blinking, especially on exposure to a bright light. Although hysterical ptosis and hysterical blepharospasm are generally present together, either may be present alone, and the proportion of one to the other may be different in the two eyes (Fig. 15).

In total blindness due to severe bilateral optic atrophy the eyes are kept open during the day and look straight forward, but they close during sleep. An uneducated man, however, if told to pretend that he is blind, generally closes his eyes, and when they are forced open or he is told to open them, he at once turns them upwards in order to keep the pupils covered by the lids. Conversely, if he is for any reason unable to open his eyes, he is likely to imagine he is blind. Hysterical ptosis and blepharospasm are thus often accompanied by hysterical amblyopia, the patient having so convinced himself that he cannot see that he makes no attempt to look when his eyes are at last opened. When the lids are forced apart, the eyes generally turn involuntarily upwards to protect them from the light. The pupils are consequently still hidden, and vision is impossible. Even when the lids are sufficiently separated to expose the pupils, the patient can see only indistinctly, and in rare cases he cannot see at all. The indistinct vision is due to a combination of hysterical paresis and hysterical spasm of accommodation. A man with normal vision relaxes his ciliary muscles to look at the distance, and contracts them to look at a near object. A man who is convinced that he cannot see, fails to regulate the activity of his ciliary muscles correctly when he opens his eyes. Instead of relaxing them when he wishes to look at a distant object, he throws them into spasm, and he also fails to see near objects clearly, as he

irritation it caused drew their attention to their eyes and resulted in blepharospasm and amblyopia, which persisted long after every sign of conjunctivitis had disappeared. These early cases were the only ones in which hypnotism was used

*Hysterical blindness following exposure to a shell explosion*—The patient, aged 22 was looking over a parapet at Gallipoli on July 18th, 1915 when a shell struck the sand bags in front of him. He remembers the sand being thrown up into his eyes after which he fell back and knocked his head. He was unconscious for twenty four hours. His first impression on regaining consciousness was extreme irritation in his eyes. He tried to open them but found he could not do so. He felt convinced that he would never be able to open his eyes or see again. The condition of his eyes had not altered when I first saw him on September 17th, 1915. He was quite blind, and there was a constant flicker of his eyelids, which were kept almost closed. On forcibly opening his eyes they were found to be turned so far upwards that it was difficult to see even the iris. A few fragments of sand were still embedded in the conjunctiva but not in the cornea. there was no inflammation

The patient was easily hypnotised, and whilst asleep he was told that he would be able to see when he woke up. The moment he awoke the suggestion was repeated very forcibly and his eyes were held open. He cried out that he could see, tears ran down his cheeks and he fell on his knees in gratitude, as he believed that his sight had been restored by a miracle. When seen again on September 20th the external appearance of his eyes was normal and he said that he could see as well as he had ever done. There had been no return of symptoms, and the patient was well in every way when I last saw him a fortnight later

I shall now describe the different varieties of hysterical visual disturbances, which were such a frequent result of gassing in the war of 1914-18. In the present war sand-storms in North Africa are likely to take the place of gas as the chief exciting cause of hysterical disorders of vision. The first case is an example of the common form in which both eyes were equally affected. The patient was unable to open his eyes owing to a combination of hysterical ptosis and blepharospasm and when the eyes were opened he saw only very indistinctly

*Hysterical blepharospasm, ptosis and amblyopia following gassing*—Gunner K. aged 33 had been in France seven months, and for most of the time was on duty behind the firing line, because his nerves were not equal to life at the front. In December 1917 he was blown up and was then very shaky and frightened. He was gassed on April 9th 1918. Next morning he was unable to open his eyes. When admitted to Seale Hayne Hospital under Captain C. H. Ripman in

of the street Unshaven, unkempt and dirty, and wearing a pair of dark glasses, he came supported by his wife, while in his hand he carried a thick stick to help to guide himself Whilst wearing the glasses he kept his eyes open, but could see nothing, when they were removed, he was unable to raise the lids owing to severe blepharospasm except in a darkened room.

Treatment was commenced the same evening, and in a short time the blepharospasm was overcome, and the patient opened his eyes, but he was still totally blind *The pupils were then found to be widely dilated, with no trace of reaction to light* The flinch reflex was completely absent in both eyes Ophthalmoscopic examination showed nothing abnormal, and a diagnosis of hysterical blindness was made Explanation as to the nature of his condition and encouragement to use his eyes rapidly led to partial restoration of vision, but at the end of two hours he still stumbled over objects placed in his path After a rest of an hour, treatment was continued and further slight improvement occurred In attempting to focus his eyes he made strong contractions of the muscles of his neck, similar to those seen in the spastic variety of hysterical aphonia when the patient attempts to speak.

The next morning he was taken out of doors, and distant objects were soon recognised An endeavour was then made to teach him to focus his eyes on nearer objects, and by the evening he could read 6/24 at 20 feet The excessive contractions of the neck muscles continued, however, but by encouraging him to relax they gradually disappeared, and vision steadily improved He was still inclined to stumble over objects placed in his path, but this was merely due to inattention On November 25th he could read with each eye in turn 6/12 at 20 feet, and he could spell words printed in small type, but as he was almost completely illiterate he could not pronounce them The flinch reflex and the normal pupillary reactions to light had returned the first evening

The patient was also completely deaf in the left ear on admission, he was given instruction in listening, and at the end of a week he could hear normally When seen four months later, in February 1919, he was at work as a watchmaker and gramophone repairer and his vision and hearing were normal

Whilst a man is still dazed as a result of being blown up by a high-explosive shell, he pays no attention to any external stimuli and may be regarded as psychically blind, deaf and anæsthetic His sight, hearing and cutaneous sensibility generally return as he regains consciousness, but if there is anything which draws his attention to his eyes, the blindness may persist as a result of auto-suggestion The following is one of seven cases seen with Mr A W Ormond in 1915, in which sand was blown into the men's eyes from the sand-bags of the parapet on which the shell exploded The

paralysis. The complete recovery after a few minutes' treatment by psychotherapy, although the ptosis had persisted without alteration for a considerable time, confirmed the diagnosis of hysteria.

*Hysterical blepharospasm, ptosis associated with over-action of the frontalis and amblyopia, following gassing*—Corporal H., aged 22 was gassed on May 20th, 1918. He was admitted to hospital the same day with conjunctivitis, his eyes being tightly closed. Thirteen days later he was able to open his left eye but the right one still remained closed and he thought he was blind in that eye. Early in June he was transferred to a military hospital in England where he remained for a fortnight. He was then sent to a V.A.D. hospital, and treated with electricity and daily eye-baths with no improvement. He was admitted to Seale Hayne Hospital on October 22nd under Captain S. H. Wilkinson, with the right eye tightly closed owing to unopposed spasm of the orbicularis, but the left eye was kept partially open as a result of continuous contraction of the frontalis muscle. Vision was so indistinct that he could see only with difficulty and he was quite unable to read. No conjunctivitis was present, but at the corner of the right eye there was a scar on the skin about the size of a two-shilling piece the result of cauterisation of a naevus when he was a child. He had always believed that this had impaired the sight of his right eye. After being in France for a few months he noticed that the sight of the right eye was becoming more "blurred" and when he was gassed he was at once terrified that he would be completely blinded in this eye. He was treated by explanation and persuasion and in half an hour the orbicularis spasm of the right side and the ptosis of the left were overcome. He was then quickly trained to focus his eyes so that his vision became quite normal except for some myopia of the right eye which had always been present and was doubtless the real cause of this eye being the weaker one.

In the following case the ptosis which was present on one side spread so as to produce facial paralysis, which was complete except for the frontalis, which attempted to counteract the paralysis of the levator palpebrae superioris. The platysma was also involved, contrary to Babinaki's teaching that paralysis of this muscle in facial paralysis is a definite sign that the condition is organic. Whilst the ptosis spread to produce facial paralysis, the blepharospasm of the affected side spread to produce facial spasm.

*Hysterical left facial paralysis and ptosis right facial spasm, paralysis of right arm and both legs aphonia and dysarthria, and amblyopia following gassing*—Lieutenant B. aged 28 was gassed on April 24th, 1918. He remained quite blind and very hoarse for about six days. When sent to England on May 3rd he could see a little, and could talk fairly well. On reaching the hospital, however he could hardly open his eyes.

August 1918, he could see only indistinctly through the narrow slit which he could produce by a great effort between his eyelids by strong contraction of his frontalis muscles, and he used his hands to steer himself. A thrill caused by contraction of the orbicularis muscles was distinctly felt in the lids when an attempt was made to force them open.

The orbicularis spasm relaxed and he recovered the power in his levator palpebræ muscles in five minutes as a result of persuasion, and he was then quickly trained to see perfectly well. For a time he was inclined to blink and to let the upper eyelids droop a little, this was due to persistence of slight ptosis, caused by hysterical paresis of the levator palpebræ muscles, and not to spasm of the orbicularis, and in order to counteract the ptosis he continued to wrinkle his forehead. He made a slow but steady recovery from this habit, and was discharged from hospital completely cured a few weeks later.

In cases in which visual symptoms were more marked in one eye than the other after gassing it was found that the patient had for some reason been anxious about the vision of the former, which may, in fact, have been less acute than the other on account of an error of refraction.

*Hysterical ptosis with unilateral blepharospasm and amblyopia*—Corporal B was gassed in France in June 1918. This resulted in severe conjunctivitis. As he could not open his eyes, he was afraid he might go blind. He was particularly anxious about his left eye, as it had always been weak and subject to inflammation and styes.

In order to protect the eyes from the pain caused by exposure to light he had kept the lids closed, and when the inflammation had subsided he found he was unable to open them. If the left lid was raised, he experienced great discomfort and everything appeared blurred. The right eye was not painful, and he could see clearly with it, but in order to do so he had to raise the lid by contracting the frontalis muscle, as he could not use his levator palpebræ.

He was admitted to Seale Hayne Hospital and an hour's persuasion was sufficient to induce him to use the proper muscles and to relax the spasm of the right frontalis, but the spasm tended to relapse for a few days unless he paid special attention to it. The vision of the left eye quickly returned with re-education of accommodation.

The next case is of interest in connection with the part taken by the frontalis muscle in ptosis. It is generally stated that hysterical ptosis can be distinguished from ptosis due to organic disease by the fact that the latter is accompanied by compensatory over-action of the frontalis muscle in the effort to keep the eye open, whereas this never occurs in the former condition. In the following case, however, as in several others, there was as marked compensatory over-action of the frontalis as occurs in any case of organic

could talk with a normal voice, but in a somewhat laboured manner. The paralysis of the left side of the face and the ptosis had completely disappeared, but there was still some slight spasm of the right side although he could now open the eye and see quite clearly and there was no squint.

In the following case continuous blinking was associated with deficient vision, which was at first ascribed to disseminated sclerosis.

*Hysterical amblyopia and blinking following irritation by a lachrymatory shell.*—Private B, aged 44 was gassed in May 1916 by a lachrymatory shell. Next day he was able to carry on, but he constantly blinked and his vision became somewhat defective. The blinking and defective vision continued but he did not go into hospital until April 1917. The thorough examination of his eyes which was repeated in three different hospitals appears to have made him fear there was something serious the matter and to have led by suggestion to severer blindness, as the amblyopia and blepharospasm now became steadily worse. The case was diagnosed as disseminated sclerosis and subsequently as cerebellar tumour on account of the swaying gait which was however simply an exaggerated result of defective vision. On admission to Netley in September 1917 his vision was 3/60 in both eyes he was constantly blinking and had a staggering gait. He was too stupid for treatment by explanation to be effective but suggestion with the aid of faradism applied to his eyelids caused the blinking to stop and his vision and gait became normal for the first time for sixteen months. The next day the blinking had returned, but he again improved as a result of further suggestion and a week later he was discharged to duty.

In the following case hysterical blindness occurred in a one-eyed man. The exciting cause would probably have been insufficient to have affected him had he not been living in perpetual fear that something might deprive him of the sight of his remaining eye.

*Partial hysterical blindness following shell-concussion in a one-eyed man.*—Sapper C, aged 28 lost his left eye in 1914 as the result of a shot-gun accident. When he enlisted in 1917 the vision of his right eye was 6/6. He went to France in May 1918 to work on the railway. In June 1918 an aeroplane dropped a bomb about twenty five yards away, but he was not hit. The force of the explosion, however was sufficient to dislodge his glass eye from its socket, and simultaneously his right eye became completely blind. The total blindness lasted for only a few days, but very defective vision persisted. He also noticed that erect objects, such as telegraph poles, appeared to be distorted. He passed through several hospitals, where the hysterical nature of the condition was recognised. He was still uncured when he was admitted to Seale Hayne Hospital.

On examination there were no signs of disease in the eye. The pupil

When I first saw him on June 16th his eyes were closed, and there was a constant spasm of the whole of the right side of the face, especially involving the orbicularis palpebrarum and levator anguli oris (Fig 15 (a)) The left side of the face, including the platysma, was completely paralysed, except for the frontalis, which contracted with excessive vigour in the patient's efforts to overcome the ptosis (Fig. 15 (b)), which was present on both sides in addition to the spasm on the right side, and prevented him from seeing unless he held his left eye open with his fingers (Fig 15 (c)) When the left upper lid was held open and the right lids pulled apart, it was found that there was a marked squint due to spasm of the internal recti The masseters were tightly contracted, so that he could not open his mouth Spasm was also present in the left side of the tongue, so that when later he was able to open his mouth

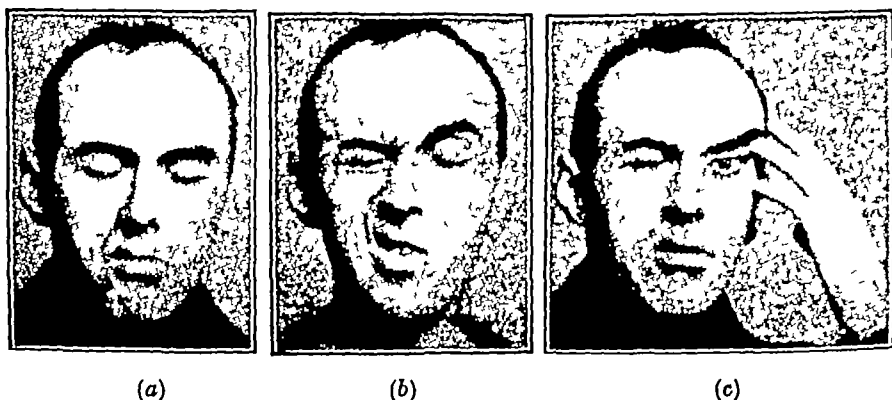


FIG 15—Hysterical left facial paralysis and ptosis, right facial spasm, (b) increased spasm with effort to overcome ptosis, (c) left eyelid raised by fingers in order to see

and put out his tongue it went to the left The right arm and leg were completely paralysed, and he was able to move the left leg only with difficulty With simple persuasion and re-education in breathing he quickly learnt to phonate, and then gradually learnt to articulate clearly The ptosis of the left eye was overcome by persuasion without difficulty The spasm of the muscles of the right side of the face slowly improved with massage and stretching the muscles by pulling the eyelids apart and pulling down the upper lip, which was gripped with one finger inside the mouth and the other outside When at last he was able to open the right eye it was found that he could hardly see with it, but with explanation and persuasion the amblyopia disappeared The severe internal strabismus, which was present when both eyes were open, gradually disappeared as he became accustomed to using his eyes With some difficulty he was taught to balance himself whilst standing, after which he quickly learnt to walk He was treated at intervals from 10 a m until 6 30 p m, by which time he could walk normally His right arm had now recovered without special treatment, and for the first time since the onset of symptoms he was able to write He

sun without blinking. The vision of the right eye was very defective but he could pick out large objects and name some of them correctly. The pupils were unequal in size the left being slightly the larger. Reaction to light was normal on the right side, but very sluggish on the left. Reaction to accommodation was irregular but was sometimes brisk in both eyes. The blink reflex was present on the right side but was completely absent on the left. Ophthalmoscopic examination showed that the retinas were normal. There was no conjunctivitis, but slight keratitis was present in both eyes, being most marked in the left.

Complete recovery followed psychotherapy the pupils became equal in size and reacted normally and a brisk flinch reflex was present on both sides. In the case of the right eye all that was required was to teach the patient to regulate the action of the ciliary muscle relaxing it for distant objects and contracting it for near objects. In the case of the left eye it was necessary first of all to convince the patient that he would be cured, and then to encourage him to look with it.

There has been much discussion in the past as to whether homonymous hemianopia can ever be hysterical. Several cases, however have been described. The following case is of interest, as hemianopia of one eye was associated with partial blindness of the whole field of the other eye.

*Hysterical blindness of one eye and hemianopia of the other*—Bombarcier U., aged 25 developed severe conjunctivitis as the result of a bombardment with mustard-gas shells on March 21st, 1918. After three weeks treatment he was able to open his eyes, but found that he was almost completely blind in the left eye, being able to see only a white mist, while with the right eye he could see imperfectly. He was admitted to Seale Hayne Hospital under Captam A. W. Gill on November 5th, 1918. The eye specialist's report accompanying him stated "left eye completely blind, right eye 6/24."

On admission there was no conjunctivitis and no sign of corneal scarring. The pupils were equal in size and reacted sluggishly to light and accommodation. The left eye was completely blind, and the flinch reflex was absent. With the right eye he could see distant objects clearly. On attempting to read with the right eye he held the book slightly to the right side so that the light from the book fell on the nasal half of the right retina, the temporal half of which appeared to be blind. The flinch reflex was abolished, except when the direction of the blow was towards the nasal half of the right retina. Peripheral vision as tested with the moving finger was extremely defective in the right eye. Ophthalmoscopic examination showed the presence of a tiny foreign body embedded in the lens of the left eye, but no other organic changes were apparent. As the result of psychotherapy complete recovery resulted in each eye both for near and distant objects and the flinch reflex was completely restored.

was of small size, and reacted briskly to light, but on attempting to focus an object he contracted the muscles of his neck, rotating his head to the right side, and alternating slight variations in the size of the pupil were evident. When he tried to read he held the book low down and either to the left or right side. At a distance of two feet from the eye he could see objects distinctly, but anything nearer or beyond this limit was blurred and indefinite. The finch reflex was completely absent. His mental attitude was one of great anxiety, and he was considerably depressed at the possibility of complete blindness.

He admitted that he went to France in considerable fear lest anything should happen to cause the loss of his one eye. The shock of the bomb explosion was sufficient to drive out his artificial eye, and this suggested to him that the temporary loss of vision resulting from concussion of the other eye would be permanent. Dr. Ida Mann thinks that the distortion of upright images was at first caused by transient oedema of the retina, which is common after concussion of the eye. Instead of disappearing with the oedema within a fortnight the distortion was perpetuated with the amblyopia as an hysterical symptom. Reassurance was speedily followed by complete recovery with return of the finch reflex.

In the next case the primary irritation of the eyes was caused by a sand-storm. It is of interest as showing two forms of hysterical blindness in one individual, in the right eye the motor element was alone involved, but in the left eye, which was more severely damaged in the sand-storm, so that the suggestion of blindness must have been stronger in connection with this eye, severe psychical blindness was present as well. The latter was almost as complete as the blindness of both eyes in the first case described, and corresponding with this the pupil of the left eye was dilated compared with the other and responded only very sluggishly to light.

*Hysterical blindness, following sand-storm*—Private F, aged 52, was caught in a sand-storm in December 1916 without any protection for his eyes. He developed intense conjunctivitis and keratitis, with severe pain and photophobia, and was unable to open his eyes. Despite the fact that the inflammation and pain gradually disappeared, he was still unable to open his eyes when he was sent home from Egypt. In March 1917, no improvement having occurred, he was discharged from the Army as permanently unfit, with a disability of 100 per cent.

On February 10th, 1919, he was admitted to Seale Hayne Hospital under Captain A. W. Gill. He wore a pair of dark glasses, on removal of which he was found to have severe bilateral blepharospasm. With explanation and persuasion the spasm of the orbicularis muscles and the spasm of convergence, which was also present, were speedily overcome. It was then found that the left eye was completely blind, so that the patient could not distinguish light from darkness and could face the

## CHAPTER XI

### HYSTERICAL STUPOR AND AMNESIA

The stress and strain of active service not infrequently result in a condition of mild confusion, which may merge into deep stupor. The onset is often gradual—a man who has hitherto been alert and efficient becomes more and more dull, slow and silent, until finally—often after an incident which has caused unusually great terror or horror—he becomes too confused to carry on, and he may wander away from his unit in a condition of stupor with more or less complete amnesia. He is then likely to be arrested and charged with being absent without leave. The majority of such cases are hysterical and their memory can generally be restored promptly by simple persuasion. Such patients should be returned to their units after a few days' rest.

Retrograde amnesia or loss of memory for the past may vary in extent and in degree. Its extent may be slight and only comprise a few hours, or it may be complete, the patient having forgotten all the events of his life up to a certain moment, which generally coincides with the moment when he came to himself. Recovery from the latter condition may be sudden and complete but in many cases it is gradual, the earliest events of the patient's life being the first to be recalled. In the case of a soldier who had been found wandering by the police and had no memory of his past life and did not even know who he was, progressive recovery took place under hypnosis. At one stage he remembered everything up to the time of his first wife's death, which had occurred twelve years before and about which he still felt acutely distressed as it seemed to him like yesterday but for some time he had no recollection of any subsequent event and did not recognise the portrait of his second wife and her children.

What is generally described as complete retrograde amnesia is the condition in which a man has lost his memory for everything that occurred before the moment when he came to himself, every thing subsequent to this moment being retained in his memory. He has no difficulty in adapting himself to his new surroundings, as he has not forgotten how to use his limbs or his voice—he can read and write and has all the elementary knowledge he acquired as a child, but anything recently learnt, such as a soldier's drill, is

The presence of a small foreign body in the left lens, which had apparently been driven in some time before, had given rise to no visual trouble at the time, beyond making the patient aware that the vision of the left eye was less good than that of the right. This no doubt accounted for the more complete hysterical loss of vision in this eye after the gassing. Believing that he was completely blind in the left eye, he appears to have subconsciously come to the conclusion that he would necessarily be blind to everything on his left side, and in this way the loss of the nasal half of the field of vision of his right eye was suggested.

#### FUNCTIONAL ASTHENOPIA, MIGRAINE

An error of refraction which has previously caused no trouble, may give rise to severe headaches in a man exhausted as the result of active service, or in a man who has been concussed by the explosion of a shell or bomb. In the constitutionally predisposed migraine may develop. The activity of the ciliary muscles and extrinsic muscles of the eyes is impaired, like that of the other muscles of the body. Doyne observed in Mesopotamia how greatly men with myopia and especially myopic astigmatism suffered from the glare. High degrees of myopia rendered men almost useless, even when wearing the glass protectors fitted with green-tinted glass which were supplied to the troops. In severe cases of concussion the patient may be unable to read for weeks owing to difficulty in accommodating. But even a slight degree of asthenopia is important if there is an error of refraction, although this may never before have caused any trouble. At first light is intolerable and the patient makes no effort to use his eyes, but after a time he begins to look at his surroundings, and this alone may be sufficient to cause headache and giddiness. Later he tries to read, the difficulties of near vision are now added to those of distant vision and his headache becomes aggravated.

The refraction of soldiers with war neuroses suffering from headache should therefore be tested if rapid improvement does not occur. Glasses must be worn all day at first; they may be given up for distant vision and subsequently for near vision when complete recovery has occurred.

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whom I was captured left me as they expected an attack. I tried to get back to the British lines but took the wrong parapet as it was very dark, and I found myself in a wood and stayed there until the next day when I met a German deserter who was making for the French lines. We found a chalk pit and there we lived as comrades for over a year behind the German lines. The deserter used to go out at night and fetch food from the camps and villages. He also got some army blankets so that we were not cold at night. I called him Fritz but had to communicate with him by signs as neither of us knew the other's language. After many months one day the German did not come back, so I had to go out of the wood myself and I noticed men walking about in khaki. In the meantime I had become confused and forgotten my name and everything else.

His memory was now completely restored and he felt perfectly fit in every way. He was discharged from the service as a man who had suffered from such severe amnesia would always remain liable to relapse when subjected to severe strain.

Some writers have advised treating all cases of amnesia, even when the blank period is very short, by hypnotism with or without the aid of hypnotic drugs such as sodium amytal but in my opinion the amnesia is protective in character and should remain untreated as we have seen several cases, in which the recovery of memory as a result of hypnosis before the patient reached our hands resulted in great distress and the development of severe tremor stammering and other symptoms which required prolonged treatment to overcome. Moreover the treatment sometimes results in the development of horrifying fantasies instead of the remembrance of genuine experiences which had been forgotten. Thus in one patient in an amytal hypnoid state genuine memories were replaced by a nightmare that during a heavy bombardment his little son was with him and got lost so that he had to search for him (Sargant and Slater 1940). An attempt should be made to restore the lost memory by suggestion under hypnosis only when the amnesia extends to periods the memory of which it is desirable to restore.

*Partial amnesia associated with terrifying dreams treated by hypnosis.*—Pte. R. an A.S.C. driver aged 24 went to France on December 1st, 1917. He remembered arriving and entraining for the front a few days afterwards, but from that moment his memory was a complete blank until December 24th, when he found himself in a C.O.S. He was admitted to Netley on December 28th. He was depressed and had an anxious expression, his forehead being deeply furrowed. In addition to the loss of memory he was troubled with terrifying dreams at night. The details were confused, but there was always a background of

forgotten He knows the use and value of money, he eats, drinks and smokes with enjoyment, and is clean in his habits He is in fact a normal individual, except that the events of his life are wiped out in every detail, so that he does not even know his name or recognise it when he hears it, and he does not recognise any person or place connected with his past life. The following case is a good example of this condition The amnesia was cured by hypnotism, which is the only treatment offering any prospect of success when spontaneous recovery does not occur in the course of a few weeks.

*Complete retrograde amnesia following prolonged solitary confinement, cured by hypnotism*—Pte W—, aged 32, was admitted under the name of J— on November 29th, 1917, to the Neurological Section, Netley He had been brought in a dazed condition by the military police into a field ambulance six weeks earlier as a "straggler" When we saw him first he was quite normal except for complete loss of memory of the whole of his past life, and he was much worried because he did not know who he was On January 14th, 1918, as no improvement had occurred and as we were unable to find who he was from his regimental records, we decided to hypnotise him He was easily hypnotised, and was asked questions, which he answered in writing He told us where his parents lived, and that he had joined the Army in September 1901, being called up from the reserve on August 5th, 1914 He was given the paper on which he had written and he kept awake the following night thinking over these things The following morning he said he could picture his home and he drew a plan of the village he came from, but otherwise there was no improvement in his memory On January 16th he was again deeply hypnotised and answered questions by writing as before This time he stated his name was W— and not J—, and that he had adopted the latter name together with the number and regiment which he found on his field card. The same day he wrote that he had fought at Mons, where he had been wounded, on the Marne, at Ypres and at "Plug Street," where his Company Commander, Captain P, was killed He was hypnotised on two subsequent occasions and further facts about his relations and friends were obtained, all of which were verified As there was still a blank in his memory from July 1st, 1916, the first day of the battle of the Somme, to November 30th, 1917, when he was found by the military police in France, he was again hypnotised and cross-questioned as to what occurred during this period The following extraordinary story was elicited, and from the fact that he was missing during the whole of the period concerned there is no reason to doubt its truth As on the previous occasions he only remembered the events described after he had studied for two or three days what he had written under hypnosis "I was taken prisoner by the Germans on the first day of the battle of the Somme I was kept in the front-line trenches until a salvo of shells came over, and the Germans, by

trunk and limbs constantly occurred. His knee-jerks were brisk and there was an extreme degree of pseudo-ankle clonus. Two days later it was noted that his knees and ankles were stiff and that his legs, hands, and face were anæsthetic and analgesic. The plantar reflexes were flexor. When he reached England on March 2nd his expression was apprehensive and he started at every sound both when awake and asleep. In his dreams he saw the ghosts of Germans he had bayoneted come to take revenge on him. He was still unable to speak, but he answered questions by nods and signs and in writing. He was able to walk with assistance. He was treated by hypnotism, but his physical and mental condition rapidly deteriorated, except that the hallucinations disappeared.

I saw him for the first time in December 1916 eleven months after the onset. He was still unable to speak. All four limbs were now completely paralysed. An extreme degree of contracture was present the legs were rigidly extended with the feet plantar flexed the arms were extended and the fingers tightly clenched though the metacarpophalangeal joints were extended. It was almost impossible to produce any passive movements but the contractures were entirely hysterical as they relaxed completely under an anæsthetic and during sleep. Total anæsthesia and analgesia of the whole body including the conjunctiva and cornea, were present except that passive movements at the elbow were painful and he occasionally suffered from toothache. The sensibility of the bladder and rectum appeared to be present as he retained perfect control over them. The fields of vision were much contracted, but hearing was abnormally sensitive and the auditory motor reflexes were extremely brisk. The conjunctival, corneal and all skin reflexes were absent.

On December 15th, 1916 vigorous suggestion with the aid of an intralaryngeal electrode during light etherisation restored the power of whispering. It was then found that he had total loss of memory he had no idea who or what he was, he did not realise that his anæsthetic legs belonged to him, and he had no knowledge of the meaning of words.

During the following months he learnt to talk a kind of pidgin English, but the meaning of every word had to be taught, and he used each word in his limited vocabulary for a variety of meanings. All forms of drink were "tea," and when petrol was poured into the tank of a motor-car he was in he called out "table has tea" table being for some obscure reason the name he applied to all vehicles. Hand represented a hand and a glove, and "to hand" was to hit. A word taught by other patients in fun would never be given up so that all forms of meat and fish and chicken were called "puss." His only numbers were one and six which represented anything more than one except a very large number which was sixty-six, or a still larger number which was six-sixty-six. The sight of his own face in a mirror always terrified him he did not realise who it was he saw but said, as he turned his head away "No like you, chuck." On seeing in the

transports rumbling along a road. Shell holes and dead bodies were seen and shells heard whistling. He had no waking memory of having been at the front, and so far as he knew when awake he had never heard a shell or seen a shell hole, but his dreams were so vivid that he could reproduce the sound of an approaching shell with great accuracy. As he did not improve with rest and occupation he was hypnotised. He went under easily, and it was then suggested to him that he should cease to worry, that the future would again appear hopeful to him, and that he would sleep well and not dream. His dreams at once became less vivid and ceased to terrify him, and he was no longer afraid to go to sleep, as he had been previously. He looked happy and the wrinkles disappeared from his forehead. He was hypnotised four times altogether, the treatment being then discontinued as he was now quite fit. We did not try to elicit from him what had happened during the "missing" three weeks he had spent in France, because he was quite satisfied not to know more, and the events were doubtless very distressing, so that the amnesia may be regarded as protective. He remained under observation for a month and had no return of his old symptoms.

The following case is the only one I have seen or heard of in which the amnesia could correctly be described as complete, the patient having not only forgotten his past history, but everything else, including the use of his limbs and speech. He could hear, but not understand, he could see, but had no notion of the meaning of what he saw. He had no sense of taste or smell and had no perception of painful, tactile, or thermal stimuli. His mind was in fact a blank. Prolonged treatment by hypnosis before we saw him had proved of no avail, but re-education led to the gradual development of a new personality, every idea having to be taught afresh as in the education of an infant. It was only after twenty-two months that spontaneous recovery of memory took place.

*Total amnesia with hysterical paralysis, contractures, analgesia and mutism due to emotional strain, recovery of memory after twenty-two months and from paralysis after twenty-eight months*—Pte M, aged 23, a basket-maker in civil life, with no personal or family nervous history, joined the Army in 1913. He was quite fit until February 19th, 1916, when he had to be forcibly prevented from going over the parapet to attack some German mortars which were firing at his trench. He then became dazed, and on reaching the aid post he could not answer questions, but he obeyed simple commands such as to put out his tongue. He believed he was still in the trenches, which were being heavily shelled, his eyes were fixed on imaginary trench-mortar shells coming towards him. His pupils were widely dilated, he sweated profusely, and his pulse was 140. Convulsive tremors of the head,

arm, which had hitherto remained rigid and paralyzed had improved so much that he could write long letters brush his hair and feed him self, but all movements were stiff, shaky and slow. The left arm improved at the same time but there was still no recovery of voluntary power in the legs. By May 31st he could stand with very little assistance and could perform all ordinary movements with his arms, though some rigidity was still present. On June 2nd, his twenty fifth birthday he stood without support, and after being helped for a few minutes he walked without assistance round the quadrangle after having been paraplegic for twenty-eight months. His physical condition now steadily improved, and by June 20th he was able to take charge of the basket making shop.

The sensibility of his skin and mucous membrane slowly returned without special treatment, but was still somewhat deficient in August. The superficial reflexes returned *passu*. Thus the abdominal reflexes were absent so long as anaesthesia was complete.

By November 1918 recovery was complete. The last trace of contracture of the fingers and of unsteadiness in gait had disappeared. The patient had regained his weight and was in every way as fit as when he first joined the Army. In March 1930 he wrote to me to say that he had remained well since I had last seen him in 1919.

Amnesia occasionally follows recovery from some other hysterical symptom, when the latter is cured by suggestion under anaesthesia or hypnosis.

*Amnesia following recovery from deaf mutism*—Sergt. H. a New Zealander was admitted under my care on September 25th, 1916. He was completely deaf and dumb and wrote the following history of his case. About 17th September our brigade was in support behind F—. A shell landed on our cookhouse, killing one and wounding two others, and as the Germans were bombarding very casually I thought there was time to get those fellows in. I had just got to them—the cookhouse was 50 yards in rear of the trenches in the open—when the enemy opened battery fire and violently bombarded the sector with large shells. My man whose both legs were broken, made progress very slow and another shell landed within six feet of us, a piece of it hitting me in the back. I remember getting to the dressing station somehow or other and next found myself in E— four or five days after. I could not walk, talk, or hear. I have all other faculties and can understand and think clearly. I am just beginning to walk a few steps at a time.

On October 1st as there was still no return of hearing or speech, he was given a little ether and after a good deal of persuasion his speech and hearing were restored. When he regained consciousness he could hear and speak perfectly but he believed that it was May 25th 1915 and thought the boys were kidding him when they told

mirror the face of the Sister who was standing by his side, he was greatly amused and said "see six sisters" All attempts to teach ideas of time, space and colour failed, and he did not recognise any of his relations, even when his father was brought to him in the middle of the night, in the hope that he might know him at the moment of waking He remembered recent events and called people by names which he invented himself a bald patient was "no-haired chick," and men who limped badly were "no-legged chick" and "six-legged chick"; all officers in uniform were "Major" and civilians "Mr" or, if friends, "Mr Chick"

In spite of treatment no improvement in the condition of his limbs had occurred by October 1917, though he was able to sit up in a chair and enjoyed being taken out of doors He delighted in childish toys, and in a general way his mind was that of a year-old child He was quite happy, but he was becoming very emaciated, as it was difficult to persuade him to eat, as he had completely lost the senses of taste and smell, and he apparently never felt hungry

On November 22nd, 1917, for no obvious reason he had a headache and became excited in the evening His memory began to return during the night, and he talked incessantly The next day he realised the deficiencies in his speech and wished to have them corrected When told a word he repeated it correctly and remembered it, and he began to form proper sentences On November 24th I cured a man suffering from hysterical aphonia with a laryngeal sound in his presence Though this had failed on many occasions since he learnt to whisper nearly a year before, it now cured him instantaneously to his intense delight He felt something snap in his head, and immediately afterwards he talked quite normally, and the memory of his home and his past life flowed back His father came the next day, and he knew him at once He soon remembered his experiences in France, but his life in the hospital was a blank, as it seemed to him that he was in France only a few days instead of twenty-one months before He had a vague recollection of very recent events, and he knew the men in the ward, but did not remember friends who had gone out only a week before He remembered "feeling funny with a buzzing head," then "something in his head was suddenly relieved," and the buzzing stopped when his memory returned

Complete anaesthesia and analgesia were still present, and cutaneous, conjunctival and corneal reflexes were absent With his eyes bandaged the only difference he could recognise between hot and cold water, tea and beer was that the tea was "sweet" and the beer was "bitter" Salt was recognised as a powder, but without taste Jelly was recognised as it disappeared so quickly, but butter was also taken to be jelly Bread was indistinguishable from fish, and he could not recognise any difference whether it was spread with butter, mustard or salt

His mental condition was now perfectly normal, but improvement in the condition of his limbs was very slow By April 1918 his right

## CHAPTER XII

### CEREBRAL AND SPINAL CONCUSSION

In the present war the majority of cases of cerebral concussion among soldiers have resulted from motor-cycle and other accidents differing in no way from those occurring in civil life. In the war of 1914-1918 the majority were the result of exposure to the forces generated by the explosion of powerful shells in the absence of any visible injury to the head, and the similar effect of bombs has been frequently observed in the last two years.

There would have been no objection to the term 'shell-shock' which was so much used in the last war if it had been reserved for the condition which follows exposure to the forces generated by the explosion of powerful shells or bombs, now generally referred to as blast, in the absence of any visible injury to the head or spine. In such cases there is an organic basis, which consists of the more or less evanescent changes in the central nervous system resulting from the concussion caused by aerial compression to which is often added concussion of the head or spine caused by the sandbags of a falling parapet or by the patient being blown into the air and falling heavily on to his head or back. On this organic basis hysterical or anxiety symptoms are often superposed.

The aerial compression generated at the moment of detonation of a high-explosive shell may amount to ten tons to the square yard which is followed by an equally great decompression, and the physical effect of bombs is still greater. This may be transmitted through the cerebro-spinal fluid to all the neurones of the central nervous system the concussion of which results in temporary loss of function, due probably to slight and temporary but none the less definite, changes in the central nervous system. The sudden violent dispersion of the cerebro-spinal fluid may rupture the delicate lymph-channels and injure the adjacent tissues. Post-mortem examinations on men who died without regaining consciousness after being blown up by high-explosive shells or bombs in the absence of any external injury showed multiple punctate hæmorrhages in the white matter of the brain, especially the corpus callosum, internal capsule and cerebral peduncles and the nerve cells especially of

him he was in England, as he had no recollection of having left New Zealand and imagined he must be in the Porarua lunatic asylum

His general health rapidly improved, and in a few days, with encouragement and re-education, he learnt to walk quite well. But the blank in his memory persisted, and at the beginning of December, when he returned to duty, he was still unable to remember anything between May 25th, 1915, and October 1st, 1916, which appeared to him to be consecutive days, and he had to learn his drill again, as he had no recollection of his military training

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of emotional strain without any concussion following the actual explosion of a shell and the automatism is also indistinguishable from that of epilepsy. The stupor following emotional strain is hysterical, it comes on gradually and is never accompanied by any sign of organic disease and it is not followed by severe head ache (*vide* Chapter XI, p. 117).

The duration of stupor varies from a few minutes to several days but it rarely lasts more than a week. It may suddenly pass away the patient having no recollection of what occurred between the onset and the moment of recovery. More frequently improvement is gradual. Some patients remain lethargic for a long time and take no interest in what is happening around them, but they may obey simple commands, such as to put out the tongue, but only after a considerable latent period. Others become child like, and I saw numerous cases, in which the patient's condition had so closely resembled dementia præcox that he was sent home with that diagnosis, although complete recovery eventually occurred.

As a rule the patient soon recovers his memory up to the time of the incident which caused it. He may for example remember the sound of a shell coming but from that moment his mind is a blank. Less frequently his memory is perfect up to a certain date such as the day of his arrival at the front but all subsequent events are forgotten. He may live over some of the forgotten events, especially those of a terrifying nature, in his dreams, but on waking he remembers nothing of them. In severe cases more or less complete retrograde amnesia may be present. But although the patient does not remember his name and has no recollection of his past life, faculties which have become automatic as a result of years of practice are not forgotten, as he is still able to read, write, talk, dress and feed himself.

The return of memory may be hastened by anything which tends to recall a man's previous occupation. A man showed great aptitude for the task when given a pair of scissors and told to cut the hair of another patient. he said that the occupation seemed familiar and it eventually turned out that he had been a barber (McDougall). A man who is fond of music may remember tunes and words of songs, especially if they are begun for him long before he remembers anything else. This is due to the fact that the singing of a familiar tune can normally be continued without any effort of consciousness after it is once started, knowledge which

the cardiac and respiratory nuclei of the medulla, showed chromatolysis with eccentric nuclei.

Several French observers found in the last war that if a lumbar puncture was performed on a man who had been concussed by the explosion of a shell within a few hours of the onset of symptoms, the cerebrospinal fluid was generally under increased pressure and contained albumin, blood and slight excess of lymphocytes. If the examination was repeated in forty-eight hours, these abnormalities were no longer present. The cerebro-spinal fluid was consequently almost always normal when the lumbar puncture was performed at a base hospital.

In such cases it is clear that organic changes occurred in the central nervous system, which were, however, so slight—consisting probably of minute capillary hæmorrhages and chromatolysis of nerve cells—that they rapidly and completely disappeared.

### Symptoms.

(a) *Cerebral Concussion* —The symptoms of uncomplicated cerebral concussion caused by blast are identical with those of concussion in civil life. The immediate effect of a high-explosive shell is to render a man unconscious, but death may be instantaneous. In the severest cases breathing is stertorous, and death may occur after an interval of a few hours or days without consciousness being regained. In the more serious of the cases in which recovery ultimately takes place the patient passes into a condition of stupor. He is at first entirely unconscious of his surroundings. He appears not to see or hear, he cannot be induced to speak, and pinching his skin produces no response. The reaction of the pupils to light is impaired or lost. He does not ask for food, but chews and swallows whatever is given him. He lies inert, and involuntary micturition and defæcation are frequently present during the first few hours. Complete insensibility is often followed by a dazed condition, in which automatic complex acts may be performed. A man may be found several miles away from his unit, but he will never recall how he covered the distance.

Many cases of this sort reach hospital diagnosed as “acute confusional insanity,” and it is not discovered until some days later when the patient’s memory had returned that the condition was the result of “blast.” It is, however, always necessary to obtain confirmation of the patient’s statement from his unit, as very similar symptoms, which were often labelled shell-shock, occur as a result

impossible because of the sense of weariness it causes. The patient finds it difficult to make up his mind even about trifling affairs this and the inability to use his brain for any length of time are often a great source of worry to him

Mental irritability is very common especially among officers and the better educated men. It is an early symptom and often continues after the patient is otherwise well. He loses his temper for trivial causes and may get himself into trouble for insubordination.

A feeling of great fatigue is often present after exposure to blast. The patient is unable to exert himself either physically or mentally. In severe cases he likes to lie like a log in perfect silence and a darkened room with his mind completely blank. It is only when improvement begins that he desires cheerful surroundings, but even then thinking requires an unpleasant effort.

Vertigo generally transient and brought on by changes in posture may occur. It is often associated with deafness or other signs indicating that it is the result of injury to the vestibule. The patient may experience momentary loss of consciousness which he describes as a black-out. Symonds (1942) has drawn attention to the occurrence of anaemia as a sequel of cerebral contusion. Though it is the commonest physical sign of organic injury it occurred in only 76 of his series of 1020 cases of head injury. The patient who has hitherto had a normal capacity for smell and has a normal nasal mucosa is unable to smell or taste. This may be complete, but is more often partial. It was associated with radiological evidence of fracture of the frontal bone in 26 and of the occipital bone in 12 cases.

Lumbar puncture seldom reveals any abnormality unless performed very soon after the injury. As it may give rise to severe headache Symonds recommends that it should be omitted from the routine investigation. The same is true of air encephalography. On the other hand Denis Williams (1941) has found that the electroencephalogram is abnormal in about half the cases so that electroencephalography repeated at intervals of some months may provide useful evidence of progress towards complete recovery.

(b) *Spinal Concussion* —Spinal concussion is generally due to an explosion in which the patient is buried under earth or sandbags. The skin and muscles over the spine may be bruised, but often no sign of injury can be found. I saw several cases in which

appeals particularly to the emotions, such as music, being most deeply engraved in the mind.

In the following case the lost memory was brought back by a fortunate accident.

*Complete amnesia, recovery on seeing name in print*—A man in an R F A uniform, but with no papers or identification disc, was found wandering by the military police. He had no recollection of his name or of any past events. In spite of numerous attempts to restore his memory under hypnosis it remained a complete blank. He appeared to be well educated and remembered everything that had occurred since he was brought in by the police. One day, six weeks later, he saw the portrait of "Madame R—, the famous Spanish *prima donna*," in the *Tatler*. His memory instantaneously returned, as he recognised Donna as his name. Subsequent enquiries from his unit confirmed his story that he had been blown up by a shell.

Symonds (1942) found that 77 per cent of 210 men whose amnesia following head injury lasted less than an hour became fit to return to full duty, in contrast with 52 per cent of 143 whose amnesia lasted more than seven days. But whilst one patient with amnesia of two or three weeks might be back at duty within four months and succeed, another with amnesia of less than an hour might not get back to duty at all or, having done so, might fail.

Contrary to common belief headache is not invariably present, Guttman (1943) found it in only 50 per cent of 181 consecutive cases. Whilst the patient is still dazed, his head feels heavy and uncomfortable, but severe pain develops only as his mind becomes clearer. Long-continued headaches are much more common after slight injuries than after severe ones, probably because the patient is unlikely to have sufficient rest after the former, and perhaps too because the amnesia following the latter is protective and saves the patient from terrifying memories. The headache is increased by the smallest mental effort, and often by even slight physical effort, such as walking. It may be brought on by bright light and by noise. It is sometimes worse at night, when it may prevent sleep. It is aggravated by nightmares and the recollection of horrors through which the patient has passed. It varies in character and position, but it is most often in the occipital region and back of the neck. For many months after the severe headache has disappeared, a heavy, full sensation may still be caused by mental concentration or excitement, and sustained attention is

cord which supplies the hyperæsthetic zone. In cases of this sort there may be some spinal rigidity, and pain may occur on bending or twisting. The x rays show no actual injury to the spine. In one case well marked striated atrophies were present on the left of the lumbar spine and to a less extent on the right, although no bruising was present, the patient had been lying flat on his back for seven months after being burned but learnt to walk the day after his admission to Netley.

In many cases feces and urine are at first passed involuntarily. The initial incontinence does not last more than a few hours. It may then be replaced by retention for twenty four hours, and this is sometimes followed by difficulty in micturition for two or three days, but the condition of the bladder was generally normal by the time the patient reached England.

#### Treatment

Complete rest in bed is the first essential in the treatment of concussion of the brain or spinal cord. In civil practice it is a common event to see patients suffering from chronic head ache after cerebral concussion and chronic backache after spinal concussion, both of which might have been prevented by sufficient rest immediately after the injury. The same is true after exposure to blast and after burial, and many men who passed from one hospital to another for many months receiving all sorts of treatment with little or no benefit, could have been rapidly cured by simple rest immediately after the injury. On the other hand, the rest must not be too prolonged and it requires considerable experience to judge the right moment when such patients should be allowed to get up. If kept in bed too long, they are liable to lose their power of standing and walking, as the care taken to keep them at rest may give them the idea that they are severely injured and hysterical astasia-abasia or paraplegia develops. In order to prevent this the patient should be made to get up to go to the lavatory and to have a bath from the first day except when severe stupor is present, in which case he should get up as soon as it has sufficiently diminished. When the patient no longer complains of pain in the head or back he should be encouraged to take exercise the amount of which should be steadily increased. Massage often aggravates the pain, and it often increases the tendency to introspection.

The patient should have the cause of his symptoms explained

concussion resulted from an actual wound, in which the missile passed near the spine without actually injuring it. Thus in one severe case a bullet entered the apex of the right lung, traversed the whole of the right side of the chest, producing a pneumo-hæmothorax, and emerged on the right side of the lumbar spine. Severe paraplegia resulted although the spine was not touched. In a case described on page 132 cervical hæmatomyelia appears to have resulted from the concussion caused by aerial compression due to the explosion of a shell without any direct blow on the spine.

Roselle and Oberthur examined a number of men suffering from spinal concussion whilst still in the trenches within a few minutes of being injured. They found that the tendon reflexes were exaggerated and the cutaneous reflexes were absent, except the plantar reflex, which was extensor, extreme hypotonus of all muscles was present. In slight cases the hypotonus passed off in a few hours, often whilst the patient was still unconscious, the legs becoming slightly spastic with normal or exaggerated knee-jerks and the plantar reflex absent or flexor. In more severe cases the muscular tone is diminished for a longer period and an extreme degree of flaccidity may persist. The knee- and ankle-jerks are then weak or unobtainable. In the course of time the tone returns, the jerks become normal, and the paralysis disappears.

When, however, more profound changes have been produced in the cord the flaccidity is replaced by increasing spasticity with increased jerks, ankle clonus, and extensor plantar reflexes. In most cases complete recovery with disappearance of all abnormal physical signs occurs, but sometimes slight spasticity with exaggerated jerks and occasionally extensor reflexes persists, the concussion having resulted in some permanent lesion of the spinal cord.

Partial or complete anæsthesia or analgesia may be present over an area of varying extent. In mild cases only the feet or legs are affected, but in the severe cases in which the knee-jerks are absent diminished sensation may extend as high as the area supplied by the spinal segment, which received the severest concussion, and there may be a girdle of increased sensitiveness to pain at the upper limit of the area of diminished sensation. In most cases the spine is tender, and in severe cases the tenderness is localised to the spine corresponding with the segment of the spinal

loss of memory may be regarded as protective. The harm which may result from restoring the memory under such circumstances is shown by the following case, which is only one of several similar cases I saw.

*Tremor and stammer following restoration of memory*—Pte K. aged 22 was admitted into a casualty clearing station in France having been found wandering and unable to give any account of himself. He did not know his name and had no idea what had happened to him. The same day he was hypnotised, and his memory for all but recent events was restored. In spite of this he continued to be given hypnotic treatment for over two months and every detail of the forgotten horrors he had recently witnessed as well as of painful experiences in his past life were daily recalled to his mind. This soon resulted in the development of a severe stammer and general tremor although neither symptom was present when he was first admitted to hospital. When he reached England it was found exceedingly difficult to control these symptoms, but with rest, isolation, re-education and encouragement recovery eventually occurred.

Active treatment is necessary only when there is more or less complete retrograde amnesia, which shows no signs of disappearing after the patient's general condition has improved. Hypnosis is the only treatment which has any effect. Three cases of complete amnesia gradually recovered after I had induced the patients to write their names and other details during hypnotic sleep. Great perseverance is generally required to restore the memory. Sometimes whilst the patient is hypnotised he describes forgotten facts or events, which at first mean nothing to him when he is awake, but in such cases the memory gradually returns.

### Hysterical Symptoms following Concussion

In uncomplicated cases of concussion recovery often occurs with remarkable rapidity. The majority of slight cases never reach a base hospital, and some indeed never go sick at all. When severe symptoms develop especially after spinal concussion, they are often the result of hysterical manifestations becoming grafted upon the organic changes caused by concussion (Hurst and Symms, 1918). Post-concussion hysteria is particularly likely to develop in men already suffering from exhaustion following the stress and strain of active service and from anxiety connected with their private affairs. It is often aggravated by a subconscious desire to evade further service in the Army. These predisposing factors make a man abnormally suggestible so that the symptoms result-

to him in language suited to his intelligence. He should be reassured about their outcome and the fear of insanity, which is often present, should be dispelled. He should be warned that such symptoms as headache, dizziness and difficulty in concentration may be slow to disappear, but disappear they will, and he should eventually be able to return to his old occupation. He should therefore make the best of things, a spirit of optimism being a great help to speedy recovery.

The patient should be sent to a special treatment centre as soon as possible after the onset of symptoms, as nothing does more harm than elaborate investigations frequently repeated, which give the patient the impression that his medical officer is in doubt about the nature and treatment of his illness. In the last war many cases reached us at Seale Hayne Hospital only after many months spent in a succession of hospitals, in each of which a more or less complete investigation was carried out, which was rarely followed by any systematic treatment. It is sad to read twenty-five years later that even now these obvious precepts are often "heedlessly and harmfully flouted" and that "the damage done by ill-advised treatment in some of these men could not be put right by a demigod" (Aubrey Lewis, 1942).

As soon as possible the patient's day is mapped out for him with periods for occupational therapy, physical exercise and indoor and outdoor games. In the early stages he should have spells of compulsory rest, and throughout should be given some, but not too much, free time in which he can occupy himself in any way he likes. It is essential that the right kind of occupation should be chosen for each individual according to the personal tastes, physical fitness at the time, and the occupation, whether in the Services or in civil life, to which he is likely to return. His psychological difficulties should be discussed from time to time, without this rehabilitation is likely to be delayed or incomplete.

As a rule no special treatment is required for the stupor and amnesia. The stupor disappears spontaneously, and in many cases complete recovery from the amnesia follows. When partial amnesia persists, it is generally unnecessary to make any attempt to restore the lost memory. In most cases the blank in the memory is for a comparatively short period, and as the events which occurred during this period were probably very unpleasant and perhaps extremely horrible or terrifying, they are best forgotten, and the

cases the injury may be fatal although no external wound can be discovered. It is thus impossible to give an accurate prognosis so long as well marked organic signs are present. As, however complete or almost complete recovery occurs in the vast majority of cases, the prognosis can be regarded as good and an attempt should be made as soon as the initial stupor has passed away to persuade the patient to walk. It is extremely difficult so long as any organic signs persist to judge to what extent the symptoms are organic in origin, but experience has shown that some physical signs of organic disease, such as an extensor plantar reflex, greatly exaggerated knee-jerks and true ankle-clonus, and unilateral absence of the abdominal reflex, may still be present when the character of the gait and its rapid improvement with persuasion and re-education show that the symptoms are almost entirely hysterical.

*Hysterical paraplegia following spinal concussion.*—Pte P., aged 28 was burned on September 25th, 1916 as a result of the explosion of a shell. The falling parapet struck him on the back, and he was dug out six hours later. His legs were completely paralysed, and he could micturate only with a great effort. He remained in this condition until he was transferred to Netley on May 18th 1917. The muscles of the legs were well developed and were not completely paralysed, but a great effort was required to contract them, the attempt leading to clonic convulsive movements. The knee-jerks and ankle-jerks were normal, and the plantar reflex was flexor. On the day of admission his attention was distracted by making him count and look at the ceiling at the same time as he clasped his hands and pulled one from the other. His legs were alternately flexed on his abdomen until the movement became quite easy. Less and less assistance was given until he was finally able to move them quite well by himself. He was then made to balance himself and repeat walking movements whilst sitting up with his legs hanging over the edge of the bed. About five minutes after the commencement of treatment he was able to stand. He next began to walk, at first with assistance, and finally at the end of another ten minutes without any assistance at all. For a time he was very unsteady but at the end of half an hour he walked round the ward. The rapid partial recovery which was produced by vigorous persuasion, was followed by slow but steady improvement with re-education. As happened in our earlier cases of spinal concussion of long standing the gait continued to be somewhat stiff and he was not fit for light duty until September. At first he complained of a considerable amount of pain in the back, but this gradually disappeared as walking improved.

*Hysterical paraplegia with organic hemiplegia caused by blast*—Pte.

ing from concussion are likely to be perpetuated and exaggerated by the suggestive effect of unskilled or too frequent investigations and unwise treatment.

In discussing the treatment of patients recovering from the effect of head injuries Rowland Hill (1943) says that he begins with the question, "Do you feel the same man that you were before the accident or have you never quite fully recovered from it?" It is difficult to think of a question more likely to suggest the hysterical perpetuation and aggravation of symptoms. What the patient requires is reassurance, and an early return to work either in the Army or in civil life after a period of training. A spirit of optimism and work with sufficient periods of recreation will make many men, even if they are not quite as fit as they were before the accident, adapt themselves to the new condition and ignore their disability. This would be impossible were Rowland Hill's treatment adopted. If a man is able to return to his previous work and do it efficiently and willingly in spite of minor residual symptoms, as many do, what possible advantage can there be in reminding him of the latter?

The organic paraplegia, which is the result of transient structural changes in the spinal cord produced by concussion, disappears with the return of the cord to its normal condition, it is then often replaced by hysterical paraplegia. Finding that he is unable to move his legs when he first regains consciousness, a man becomes convinced that he is paralysed and makes no further efforts, the paralysis thus produced by auto-suggestion can be removed by persuasion or a counter-suggestion. In the same way when one side of the brain has been chiefly affected by the concussion, an initially organic hemiplegia merges into hysterical hemiplegia. I have watched several cases, in which all the physical signs of organic paraplegia or hemiplegia were at first present, but have gradually disappeared in the course of a few days or weeks, although the paralysis has remained, until it has been cured in a few minutes by simple psychotherapy. Sometimes, however, some organic signs remain, and psychotherapy then can produce only an incomplete cure, a slightly spastic gait or some slowness and lack of accuracy in the finer movements of an arm being left as the permanent result of the concussion. Less frequently the original changes in the central nervous system are so severe that the symptoms remain organic in origin throughout, and in exceptional

headache had disappeared and the pain in the limbs was less marked. By May 22nd the muscular tone had improved the knee-jerks were now increased, ankle-clonus was elicited on the right side and the plantar reflex was extensor on the right side but normal on the left both abdominal reflexes were absent.

On admission to Netley on May 30th he could move both arms and legs but they were very weak, the right side being worse than the left. Slight pain was still present in the hands and arms but the pain in the legs had disappeared. There was marked wasting of the muscles of the upper limbs especially of the hands, the atrophy and weakness of the right hand being severe. The knee jerks were exaggerated and true ankle-clonus was present on the right side and also though less well maintained, on the left. No abdominal reflexes were obtained, and the plantar reflex was definitely extensor on both sides. The skin was much thickened over the palms of the hands and soles of the feet. It seems clear that a hemorrhage occurred into the cervical spinal cord at the time of the explosion probably as a result of acrial concussion rather than of the concussion caused by falling after being blown into the air as the patient was quite clear that the fall did not hurt him particularly and that he could not put out his arms to save himself when he was in the air.

On June 11th the patient being still unable to sit up in bed and no definite progress having been made with his power of moving his arms and legs, it was thought that some of the incapacity might be hysterical in spite of the definitely organic basis. He was therefore treated by vigorous persuasion in five minutes he was sitting up in a chair and at the end of a quarter of an hour he was able to stand and take a few steps with comparatively little support. During the next ten days he learnt to stand and walk with an almost normal gait and without assistance. His condition must therefore at this stage have been largely hysterical and due to perpetuation and exaggeration of symptoms, which were originally entirely organic and were still to some extent a result of organic changes in the spinal cord. Steady improvement followed in the hands and legs his gait became almost normal, and he could use his hands for all ordinary purposes though there was still some atrophy and weakness of the small muscles. The right plantar reflex remained extensor the left flexor and the abdominal reflexes did not return.

*Hysterical paraplegia following organic paraplegia due to a wound of the spine received seventeen months previously*—Lt.-Opl. E. aged 43 was wounded in the back by shrapnel on September 27th, 1916. He immediately became paraplegic. A laminectomy of the sixth and seventh dorsal vertebrae was performed on October 10th, and a piece of shrapnel was removed. He had incontinence of urine and constipation for several weeks. By the end of February 1917 he could get about on crutches with difficulty. He was transferred from hospital to hospital before he was finally transferred to our care at Netley on March 20th 1918. On admission he could stand only with the aid of

M, aged 25, enlisted in September 1914, and served for six months in France and a year in Salonica. He was very fit the whole time, and was never worried by the shell fire. On November 22nd 1916, he was blown up by a shell, and remained unconscious for four days with signs of complete organic left hemiplegia and incontinence of urine and fæces. He began to answer questions on December 2nd and complained of severe headache. His knee-jerks were then greatly exaggerated, especially the left, and the plantar reflex on both sides was extensor. The headache soon disappeared, and the hemiplegia gradually improved, but he was still quite unable to walk when he reached Netley on January 24th, 1917. He had no recollection of anything between the fight, in which he was blown up, and the last few days he was in Malta. The knee-jerks were exaggerated, the left plantar reflex was still extensor, but the right was now flexor, and the left abdominal reflex was absent. The inability to walk was hysterical, and it disappeared the day after admission as a result of persuasion, with further re-education he soon learnt to walk without even a limp. By February 2nd the left plantar reflex had also become flexor, and the left abdominal reflex was as brisk as the right, but Babinski's second sign (combined flexion of thigh and pelvis) was still positive and the knee-jerks were as before. The Wassermann reaction was negative. He was discharged to duty in April, the superficial and deep reflexes being normal and equal on the two sides, but Babinski's second sign was still present, though less marked. A striking feature in this case was the disappearance of the extensor plantar reflex, which had persisted for sixty-four days, within nine days of the hysterical paraplegia being cured.

*Hysterical paralysis associated with organic paralysis due to hæmatomyelia, the result of spinal concussion following shell explosion*—Pte A. C., aged 24, was in a charge on April 11th, 1917, when he heard a crash and was blown three feet into the air, falling heavily on his face. He did not lose consciousness, and he is quite certain that his head was not doubled under him. He was unable to move for several hours, except that he managed to raise his face out of the mud in order to breathe. On being taken to hospital he remained quite helpless. His elbows were kept acutely flexed, as in lesions of the fifth cervical spinal segment. His right arm and leg were completely paralysed, and only very feeble movements were possible on the left side. He had much aching and tingling pain in his limbs and a spasmodic gripping pain in the calves. He had some retention of urine during the first day, and a catheter was passed on one occasion, but after this his bladder and rectum showed no abnormality. Though listless and suffering from headache, his mind was not confused and his speech was normal. Knee-jerks were very weak, and no definite plantar reflex was obtainable. By the 20th a slight degree of power had returned in the right arm, both arms were still painful. On May 12th it was noted that although there was no anæsthesia, sensation to light touch was diminished up to the region of the clavicle. The

and had experienced no desire to micturate. During the day he could with an effort pass a small quantity of urine at a time but apart from this it continuously dribbled away and he wet his bed every night. There was at first some constipation but his bowels were now regular. He stated that his legs had felt weak for some time but there was no definite paralysis.

He was discharged from the service uncured in September 1918 and admitted to Seale Hayne Hospital on December 2nd as a pensioner. There were no physical signs of organic disease. The functional nature of the condition was explained to him and he was kept in bed for a few days. He steadily improved and by January 5th 1919 the nocturnal incontinence had completely ceased. He was still lacking in confidence during the day and at first slight dribbling occurred at times but he at once gave up his urinal and soon did not pass urine more often than every three hours.

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crutches. The right knee-jerk was greatly exaggerated with response to the lower end of the tibia and slight spread to the opposite side; ankle-clonus was marked and sustained, but the plantar reflex was flexor. The left knee-jerk was exaggerated to a less extent, ankle-clonus was present, but not so well sustained, and the plantar reflex was flexor. With persuasion and intensive re-education he was walking in less than an hour. In a month his gait was normal, but rather heavy. The signs of organic disease remained unaltered. He was then discharged from the service, but was quite fit to follow his civil occupation.

*Spinal concussion involving posterior columns associated with hysterical paraplegia*—Pte W, aged 32, was buried by a collapsing trench on July 30th, 1917, he was fit in every way before this happened. When admitted to hospital in France he was unable to speak or move his legs, and it was found that he had no knee-jerks. His speech returned in a few days after stimulation with faradism, but he continued to stammer. On admission to Netley on August 28th he was still completely paraplegic and had a severe stammer, both knee- and ankle-jerks were completely absent, and there was considerable rigidity of the legs. The plantar reflexes were normal. As a result of vigorous suggestion with the aid of faradism he was induced to walk on the day of admission, and with re-education his speech and gait slowly improved. At the beginning of January 1918, the knee- and ankle-jerks were still absent, and a slight Romberg sign was present, but he walked almost normally. The Wassermann reaction of the blood and cerebro-spinal fluid was negative, and no abnormal cells were found in the latter. It seems probable that the loss of jerks and the inco-ordination were due to the spinal concussion having involved especially the posterior columns, as in a fatal case the spinal cord of which was examined by Mott. The response to treatment by suggestion and persuasion shows that in spite of this the paraplegia was largely hysterical in origin, the paralysis due to the concussion being perpetuated by suggestion. The speech defect was of course entirely hysterical.

Three cases of hysterical incontinence and one of hysterical retention of urine following spinal concussion were admitted to Seale Hayne Hospital and described by Moore (1918).

*Hysterical incontinence, following concussion of spine, of eleven months' duration, cured by psychotherapy*—Gnr E, aged twenty-two, went to France in June 1917. In February 1918, a 59 shell exploded close to him, and he was wounded in the back immediately to the right of the middle line about the level of the iliac crest. A foreign body was removed the same day in the C C S. On the following day, when being sent down the line by train, he had retention of urine and had to be catheterised. For the next three weeks a catheter was passed two or three times daily. Incontinence of urine had been present ever since, and he had constantly worn a rubber urinal. He had had no pain

to the sights and sounds which at first frighten all but the few who do not know what fear is, the nervous system does not wear well, and unless regular periods of rest from the mental strain are given the majority of men gradually become irritable, restless, and unable to perform their duties satisfactorily.

The continuous and monotonous strain of active service in the Navy might have been expected to result in functional nervous disorders, but the fine spirit of confident superiority in the men and the excellent hygienic conditions prevented the development of neurasthenic or psycho-neurotic symptoms in any but exceptional cases (Rolleston).

The sanitation of the Army in France was so efficient between 1914 and 1918 and again in 1939 and 1940 that the second great cause of neurasthenia—acute and chronic infections—was less prominent than in any previous campaign. But comparatively slight toxæmia, such as that due to trench fever diarrhoea or tonsillitis, which is not sufficiently severe to make a man go sick may cause nervous exhaustion which would have been prevented if sufficient rest had been taken. A subaltern in the Flying Corps, who was actively engaged almost daily from September 1914 passed unscathed through all the fatigue and excitement until January 1915 when he had an attack of acute tonsillitis. He was taken to a hospital where there was a shortage of food, and he was then conveyed in a half-starved condition to England the journey occupying over thirty hours. The fatigue and insufficient food were not new experiences, but the addition of the toxæmia caused by the tonsillitis, which by itself would have only led to a few days of illness, was sufficient to cause a condition of severe nervous exhaustion the recovery from which was, however remarkably rapid. In the early months of the last war I saw several cases of neurasthenia following anti-typhoid inoculation, when the patient had to march a long distance immediately afterwards, but no more occurred after the authorities wisely issued an order that all inoculated men should be off duty for forty-eight hours.

The armies in other parts of the world between 1915 and 1918 did not share the immunity from serious epidemics with the army in France. Few men remained on Gallipoli for more than a month after August, 1915 without suffering from chronic diarrhoea or falling a victim to dysentery paratyphoid fever or epidemic jaundice. Added to this was the impossibility of withdrawing the troops from

## CHAPTER XIII

### EXHAUSTION RESULTING IN NEURASTHENIA

#### **Ætiology.**

By neurasthenia in this chapter I mean the condition of exhaustion which may result from physical and mental strain, the toxæmia of acute and chronic infections, insufficient food, excessive heat, and the pain and toxæmia of wounds

Although it would require a very exceptional nervous system to pass unscathed through twelve months or more of war under the conditions prevailing on every active front from 1914 to 1918, neurasthenia never resulted solely from prolonged physical strain. The utter nervous exhaustion caused by pure physical fatigue, however intense it may be, does not give rise to any persistent symptoms, and a few days' rest always leads to complete physical and mental recovery. This is well shown in the following description by the war correspondent of the *Daily Chronicle* on April 3rd, 1918, of men who had fought continuously for six days and nights. "They were tired almost to death, and when called on to make one last effort after six days and nights of fighting and marching, many of them staggered like men who had been chloroformed, with dazed eyes and grey and drawn faces, speechless, deaf to words spoken to them, blind to the menace about them, seemingly at the last gasp of strength. Towards the end of this fighting they had a drunken craving for sleep, and slept standing with their heads falling against the parapet. In body and brain these men of ours were tired to the point of death. They felt like old, old men. Yet after a few days' rest they were young and fresh. It was almost impossible to believe they were the same men. They had washed off the dirt of battle and shaved, and the tiredness had gone out of their eyes and their youth had come back to them." These words would describe with equal truth the men who escaped from Dunkirk twenty-two years later.

Life in the trenches is always accompanied by a state of nervous tension. Danger is ever present, and during periods of activity a man can only sleep at odd moments, and the night may bring increased responsibility and anxiety instead of rest, especially to young officers. Although most men become gradually accustomed

The physical signs of exhaustion disappear after sleep and the cells of the central nervous system suprarenal glands and liver are simultaneously restored to their natural condition unless the stimulus is very excessive, when a considerable period of rest may be necessary before complete restoration occurs. This explains how the apparently exhausted army which had not slept during the retreat from Mons, was yet able after two or three days rest to turn and defeat the Germans in the battle of the Marne.

Similar changes occur in the brain as a result of acute and chronic infections. These are seen in Orle's microphotographs of the brains of men who had died from paratyphoid fever and streptococcal septicæmia. The clinical and pathological observations of Sergeant, Loeper and Oppenheim, and others in France and of Elliott in England have made it probable that the muscular weakness and feeble circulation in certain acute infections, such as typhoid and paratyphoid fever, bacillary dysentery and malaria, are not so much due to the effect of the toxins on the skeletal muscles and myocardium as to their effect upon the suprarenal glands, the neurasthenia, which is a common sequel of these infections, being largely due to suprarenal exhaustion. This is especially likely to be the case when the glands are already in a condition of exhaustion owing to prolonged mental and physical strain. In fatal cases of malaria the suprarenals are packed with parasites and show an extreme degree of degeneration.

### Symptoms

The symptoms of neurasthenia in soldiers do not differ from those of the neurasthenia of civil life. Headache rapid fatigue on mental and physical effort, and difficulty in concentration are always present. The patient may wake up feeling fairly well but becomes rapidly fatigued and is completely exhausted by mid-day or earlier in contrast with the depression phase of the manic-depression psychosis, in which the patient wakes up tired and tends to get better as the day goes on. The appetite often remains excellent and the digestion good.

A fine tremor of the hand was common in men suffering from neurasthenia. I often noticed it in officers who had been home on leave for a few days and had regarded themselves as perfectly fit. The symptom was sometimes very persistent and was often a

the shell-swept area, for every corner on the Peninsula was exposed to hostile fire. Thus it came about that there was hardly a man at the end of November 1915, who was not suffering more or less severely from nervous exhaustion due to fatigue, prolonged mental strain and toxæmia. In Salonica the hot weather and the virulent form of malaria, for which the Struma Valley has always borne an unenviable reputation, were the cause of many cases of neurasthenia. The effect of the numerous infections rife in Mesopotamia and East Africa was aggravated by the great heat, and at times by the insufficient supply of food. In the present war the Eighth Army has been remarkably free from disease with the exception of dysentery and infective hepatitis, neither of which, however, has been of a serious character. Dysentery was much more widespread and severe and enteric fever was comparatively common among the Italians and Germans, and one factor in our success at El Alamein is said to have been the enfeeblement of the enemy brought about by dysentery.

The pain, loss of blood and toxæmia, which may result from severe septic wounds, often cause neurasthenia, especially if the soldier is already over-fatigued from mental and physical strain. The frequency of this has been greatly reduced in the present war owing to the effect of sulphanilamide compounds in overcoming sepsis.

### **Pathogenesis.**

Mental and physical strain and severe pain lead to exhaustion by their direct action upon the nervous system, and indirectly by their action upon the suprarenal glands, together with the liver and possibly the thyroid. Prolonged muscular exertion and profound emotions, especially fear and anger, have been shown to produce chromatolysis of the brain cells. This was well seen in Crile's microphotographs of the brains of rabbits and cats, which had been subjected to great exertion, acute and chronic fear, and prolonged insomnia, and of a soldier, who died from exhaustion caused by excessive physical and mental strain, hunger and thirst during the retreat from Mons.

At the same time the overaction of the suprarenal glands, which is a constant feature of excessive muscular activity, fear, anger and pain, leads to disintegration of their cells, which show loss of cytoplasm and misshapen eccentric nuclei, especially in the cortex. Similar changes, together with loss of glycogen, are found in the liver. No other organs show any changes.

complete exhaustion. He was dazed and had a severe headache. He lay motionless in bed and would not answer questions for four days. He then got up said he was quite fit again, and asked to be sent back to duty.

For the first few days the patient should not read or have visitors, but in most cases these rules can soon be relaxed, and cheerful companionship with plenty of amusement and interest hasten recovery.

Progress is much hastened by combating insomnia, as exhaustion cannot disappear so long as the patient sleeps no more than three or four out of the twenty four hours. I found barbitone (medinal) gr. v to x with aspirin gr. x the most useful hypnotic. The dose of the former can generally be reduced by one grain a night after the first two nights till no more is given, and the aspirin can then be halved and finally discontinued. In the early stages strychnine may prove useful but benzedrine makes the patient sleepless and should be avoided. On the other hand, in depressive states benzedrine is often of value but the symptoms are aggravated by strychnine.

As soon as the patient is well enough to get up he should begin to help in the ward and a few days later he should be given light employment out of doors. For—

The cure for this ill is not to sit still  
And frown with a book by the fire  
But to take a large hoe and shovel also  
And dig till you gently perspire

The amount of exercise should be gradually increased, and the patient should not be sent back to duty until he can do moderately heavy work without undue fatigue. The best results were obtained if the patient was kept under a certain amount of discipline, and for this reason we found it most satisfactory to send him direct from hospital to a command depôt where he received physical training to prepare him for a return to duty instead of to a convalescent or auxiliary hospital, where the comparative freedom from supervision and the lazy life too often made him unfit for any kind of military service. At one time I sent soldiers recovering from neurasthenia to a special convalescent hospital where they could be given progressive exercise before returning to duty. But I soon found that the complete absence of military discipline led to a

source of great worry to the patient, who regarded it as an indication of the presence of some serious nervous disease. It should not be confused with the coarse tremor of emotional origin which was often perpetuated as an hysterical symptom (*vide* p 61)

The symptoms described under the name of "effort syndrome" may be prominent (*vide* p 207) In most cases the blood pressure was low. In some cases, especially when the neurasthenia had developed after paratyphoid fever or malaria, the symptoms were exactly what might be expected to result from hypo-adrenalism, the extreme asthenia, low blood pressure, hypothermia, and feeble digestion characteristic of Addison's disease were present, but instead of becoming progressively lower, the blood pressure slowly rose as the general condition improved (Hurst, Bury and Wilkinson, 1919).

In a much smaller group of cases, all of which followed prolonged mental strain, the patient's mental and physical condition resembled very early myxœdema, and treatment of the hypothyroidism by small doses of dried thyroid gland led to rapid improvement when the condition had become stationary after the initial progress which had resulted from rest Most of the cases were sent home in company with congenital mental defectives, on account of being too slow and stupid to be of any value as soldiers, but further enquiry showed that some had been well educated, and that they had been quite normal in civil life and during the first few months at the front

The patient was often depressed and hypochondriacal, and definite anxiety symptoms were frequently present These symptoms are psychogenic and must be regarded as a complication of neurasthenia and not part of the neurasthenic state, which is physical and not nervous in origin.

### Treatment.

Complete physical and mental rest are at first essential The patient must be kept in bed until he no longer feels tired In slight cases two or three days are enough, and it is never necessary to continue the complete rest for more than a fortnight Recovery is remarkably rapid when the symptoms are due to pure physical exhaustion A private, who had fought in almost every battle since the retreat from Mons without being wounded and without having any leave, was admitted in March 1917, in a condition of

instinctive activity. So fixed, however is the primitive association of anger with fight and fear with flight that when the natural sequels of these emotions are restrained, they continue to give rise to suprarenal and probably thyroid activity.

Thus the ceaseless fear felt by the constitutionally timid when exposed to the horrors of war results in constant over-secretion of the suprarenal and thyroid glands, the physiological results of which are not followed by the muscular activity of flight for which they are the preparation. The unexpended energy may be so extreme that the soldier is incapacitated by it. On reaching the safety of a base hospital, the hyperactivity of the suprarenal and thyroid glands and the signs and symptoms to which they give rise often disappear. But they may be perpetuated by war dreams, and in severe cases the mind is absorbed by day as well as by night by pictures of the horrors which the individual has witnessed. Every sound reminds him of shells and every movement suggests the approach of danger. The activity of the suprarenal and thyroid glands is consequently maintained, and the patient presents a picture suggestive of Graves's disease although hyperactivity of the suprarenal glands is probably of more importance though less easily recognised than that of the thyroid. The pulse is rapid, especially on the slightest exertion and the cardiac impulse is heaving and diffuse but there are generally no murmurs. The patient may complain of throbbing pain in the head, dizziness and palpitation, especially on exertion. The average blood pressure in men living in areas behind the trenches and exposed to occasional shell fire but in good condition so far as food, sleep and shelter from the weather were concerned was 110 to 120 mm. systolic and 75 to 80 mm. diastolic. In front line trenches the systolic pressure rose to 120 to 130 mm. and at times of increased activity to 140 to 160 mm. with a diastolic pressure of 70 to 100 mm. (Cowell). Monard (1916) happened to be measuring the blood pressure of a man a few seconds before a shell exploded in the immediate neighbourhood. It at once rose from 125 mm. to 130 and ten minutes later was 150 an hour later it had again fallen to 125 mm. In the functional hyperthyroidism-hyperadrenalism of soldiers the blood pressure is always raised whilst the patient is at the casualty clearing station, and it is often 150 to 160 mm. and in severe cases even as high as 180 mm. on arrival in England.

I observed a characteristic cutaneous reaction in these cases, but

adrenaline independently of any sympathetic action. The central nervous system, the heart and the muscles of the limbs and trunk are thus prepared for great activity at the expense of the digestive organs, which do not contribute to the efficiency of the individual during strenuous exertion. The evaporation of sweat, the secretion of which is increased, prevents the rise of temperature, which would otherwise occur with excessive muscular activity, and the deep respiration and the relaxation of the bronchioles allow more oxygen to enter the lungs and excess of carbon dioxide to escape.

The partial asphyxia, which is the cause of the painful gasping caused by over-exertion, stimulates the secretion of adrenaline. The increased supply of sugar and of blood to the muscles, the diminution in muscular fatigue and the relaxation of the bronchioles which result are the cause of the "second wind," which makes the individual capable of renewed activity, when he is about to fall a victim to his enemy owing to being unable to continue in battle or flight after the initial secretion produced by anger or fear is beginning to wane.

Adrenaline acts very rapidly but for a short period, as it is quickly oxydised. It is consequently of special importance when an emotion calls for sudden activity. When more prolonged activity is required, the internal secretion of the thyroid gland helps to maintain the activity initiated by adrenaline, as it acts after a longer latent period and its action is more prolonged. Experimental evidence for this is lacking, as there are no biological tests for thyroid secretion, similar to those which prove the presence of excess of adrenaline in the blood, but Cramer (1928) has shown by histo-chemical methods that the activity of the thyroid gland is increased by stimulation of its sympathetic nerve supply.

It is

When the burning moment breaks,  
And all things else are out of mind,  
And only Joy of Battle takes  
Him by the throat, and makes him blind,\*

that the physiological effects of the fury and excitement of battle enable the born soldier to perform feats of strength and endurance, which may be in striking contrast to his comparatively feeble physique. But the changes which accompany fear and anger are entirely useless if the emotions are not followed by the associated

\* Julian Grenfell

from the sleek appearance when in civil life was most remarkable (Fig 16). One man who kept his hair closely cropped said his hair reminded him of the bristles of a hedgehog. The persistent action of the pilomotor nerves appears to last longer than any other symptoms, as it was still present several months after a man's return from France, at a time when he had become otherwise quite fit. In all cases it is associated with an exaggeration of the general cutaneous pilomotor reflex, though this is not often well marked and is unaccompanied by any vasomotor reflex. In some cases the hair on the body as well as on the head has been persistently erect. It is difficult to give a physiological explanation for the case of an officer whose hair turned white after he had been buried in a prone position for twenty two hours, and a seventeen year old boy who became grey after a week's heavy bombardment, although similar events have often been described in civil life.

The hands and occasionally the eyelids are tremulous, and the patient is very nervous and excitable. The eyes are often slightly prominent (Fig 17). This symptom was much more commonly seen near the front line than at the base. W Johnson found that the prominence of the eyes often disappeared within two or three weeks if the patient was allowed to rest for this period at a casualty clearing station, although the persistence of other symptoms necessitated his evacuation. The pupils are generally dilated and react slowly and to a diminished degree to light. The deep reflexes are often much exaggerated. Diarrhoea is occasionally present.

An ordinary seaman aged 18 who had already been twice torpedoed, complained of constant vomiting and inability to do any thing. His pulse-rate was 102 he had a fine tremor of hands and tongue and slight exophthalmos and sweated excessively presenting a picture of fairly severe hyperthyroidism. Though sent ashore the following day the symptoms persisted for three days (Surg Lieut J C Ryle, 1943).

The thyroid gland, though over-active, is generally not obviously enlarged, but in some cases moderate enlargement is present.



FIG 17 — Pte. E., severe hyperadrenalism and hyperthyroidism with exophthalmos, resulting from prolonged terror

in no other condition. On moving the finger over the skin of the chest so lightly that in normal individuals no local reaction would occur, a pilomotor reflex is almost instantaneously produced, goose skin is obvious, and occasionally the hair can actually be seen to stand on end over the area touched by the finger and for some distance on each side. After about five seconds the pilomotor reflex fades away and is at once replaced by a vasodilator reflex, the blush, which may have a white border on each side, often lasts for several minutes. As improvement occurs the vasomotor reflex disappears, but the pilomotor reflex generally persists for a few weeks longer. Symms and I never found this sign in its fully developed form in 100 consecutive cases of other war neuroses,

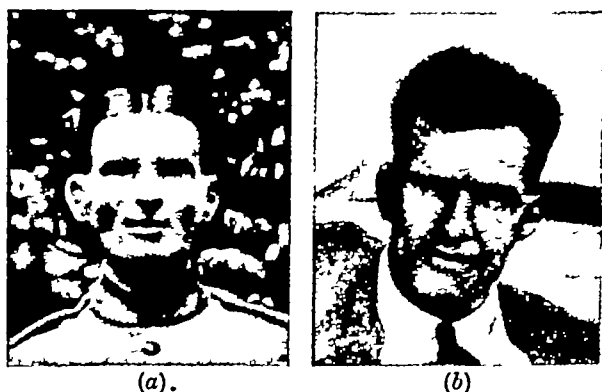


FIG 16—L-Corpl W, with hair continuously on end after prolonged terror  
(a) before going to France, (b) after being sent home

21 cases of other forms of effort syndrome, and 47 men rejected for military service on account of various diseases

The circulatory symptoms may be so prominent that the case was often diagnosed as "disordered action of the heart." The condition may then be described as the endocrine type of the effort syndrome. Excessive sweating often occurs, sometimes in paroxysms, especially over the palms of the hands and soles of the feet, and the patient frequently loses weight.

An officer told me that he felt a curious tingling sensation over a broad band of skin round the trunk every time he heard the whistle of an approaching shell, this sounds like a pilomotor reaction. I saw several men suffering from the effects of severe emotional strain, whose hair persistently stood on end and could not be kept down by means of grease. In some cases I had the opportunity of comparing their appearance with what it was formerly, and the change

## CHAPTER XV

### ANXIETY NEUROSES OF WAR

By T. A. Ross M.D. F.R.C.P.

By the word neurosis in this chapter is meant those symptoms of an illness which are mentally determined as opposed to symptoms which are physically determined. The noun neurosis has its adjective neurotic. For many people it has acquired a moral significance, which the noun has not. Here no such significance is attached. It is used simply as the adjective to the noun. If a person faints at the sight of blood it is because of some idea. That is neurosis. If he faints because he has lost a large quantity of blood the symptom has been brought about by physical happenings. These distinctions seem fairly clear yet there are those who will point out that in the first example there were many physico-chemical happenings from the moment the stimulus entered the eye till it was registered in the mind, and they will ask how do we know that these were not responsible for the faint. This may be true, but if it is then the same sort of things must be responsible for all our so-called mental processes, all of which depend either on past or present sensory impressions. The same kind of people will point out that mind and body are one, because we have no knowledge of one without the other. This is true, but even these people for practical purposes separate their mental from their bodily activities. They think that thought and the appearance of choice are mental affairs, that digesting their food is a bodily one. In practical affairs it does not answer to be swayed too much by nice philosophical considerations, and this division of symptoms into physico-genic and psycho-genic is altogether a practical one. We shall be merely foolish if we treat diphtheria by persuasion, equally so if we treat a neurotic fainter with digitalis. With all this it is admitted that the mind constantly affects the body and that physical events in the body affect the mind.

It is often easy to make the distinction and assign any given case to a neurotic or to a physical category. Unfortunately it is often difficult, not least in war cases. A soldier has had dysentery and trench fever has lost any enthusiasm he had for the war and

McNee and Dunn (1917) found that the average weight of the thyroid gland in 65 apparently healthy men killed in action was 26.7 gms, which is not above the normal for civilian men of corresponding age.

Though hyperthyroidism is the most obvious condition present, it is not the most important, as in all probability it is accompanied by hyperadrenalism, which accounts for the high blood pressure, and perhaps for the exaggerated pilomotor-vasodilator cutaneous reflex.

Considering that adrenaline gives rise to an increased production of sugar, glycosuria was a less common result of the stress of active service than might have been expected. Diabetes occurred no more frequently among British soldiers than among men of the same age in civil life (Williamson). This was, however, not the case in Germany. Among 250 diabetic soldiers treated by Lenné at Neuenahr, only 5 were predisposed by their family history and only 27 had had glycosuria before. In 95 the onset had been acute with sudden great thirst, and in many the condition seemed clearly to be the direct result of exhaustion and mental strain.

### Treatment.

The patient must be isolated at first from the other patients in the ward by screens, and should only see such visitors as he believes will allay rather than increase his nervous irritability. The best results are obtained with the same type of psychotherapy as that required for the anxiety neuroses.

Mental activity and restlessness are lessened by small doses of bromide and phenobarbitone.

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bring forth a blush or dryness of the mouth. All these are common, but as a rule they do not last long. If they do not disappear soon, if instead of lasting for minutes they last for weeks or for months so that the patient is driven to the doctor then they are called neuroses and looked on as illnesses. The doctor too often does not place them in the category of mentally caused diseases, but is apt to think that there must be some physical agency at work which is causing this prolonged departure from normal functioning.

The object of this chapter is to show that there are numbers of patients who have been ill for a long time simply because their emotional reactions have become crystallised. Why should they? The answer to this provides the cure of the illness. We can enumerate some of the reasons.

(1) The patient may be of poor psychical resistance. His history will show that he has been upset by untoward circumstances almost every time that these occur. Difficulties in life are common, and he has so many reactions every time they happen that he is often ill. He becomes unreliable. Nobody wants him, and therefore a state of chronic depression sets in with almost chronic symptoms. He may have his good days, but his bad ones keep coming back. This may be due to a poor inheritance or to a bad education, or as is perhaps commonest, to both. A nervous woman has a child, and she has hated the whole business of having it, and she declines to have another. This child having a poor inheritance should receive the best kind of bringing up but it is safe to say that it will receive the worst. The mother may be devoted to it, but it is not good for a child to receive the entire output of devotion which one woman can give it. It ought to receive only that fraction which a member of a more numerous family can get. To overwhelm a child with devotion is to make it unfit for contact with the outside world. The mother will probably be fussy about its health and have all sorts of rules for its conservation constantly in force, so that the child grows up believing that health is a precarious thing, and that unless we are always taking care of it we shall fall ill. On the other hand, she may be aware of these dangers and be quite unnecessarily harsh and callous, so that the child learns to dread all seniors and superiors from the beginning and approaches all people in authority in fear. If it be asked how anyone should bring up an only child, the answer probably is that no adult can bring up children at all. The adult mind is too remote

is presenting symptoms which might be due to a physical illness or which might be neurotic. Sometimes the diagnosis will be impossible. Sometimes it will depend more on the habits of thought and predilections of the doctor than on the objective findings. Yet we must try to make the diagnosis, for it is as damaging to a patient to make a diagnosis of physical disease when there is none as to miss the physical when it is present. There are large numbers of people living the life of chronic invalids because some doctor diagnosed a physical illness which was non-existent.

At all times of which we have record people have been aware that emotional states caused bodily results. The bible is full of it. Anorexia nervosa, pseudo-angina, diarrhoea, nausea, utter exhaustion, are all described in it as arising from mental causes. It is possible, however, that it is not realised how protean the symptoms of depressive emotions may be. headache, failure of concentration with poor memory as a consequence, difficulties of vision, all sorts of odd sensations in the head, poor sleep, bad dreams. These are some of the head symptoms which any depressing emotion can bring about. The other systems of the body are equally susceptible. In the alimentary system suppression of saliva, alteration of gastric secretion and movements, constipation or diarrhoea are all common. In the circulatory system we find fainting, palpitation, pallor, flushings, raising of blood pressure. Alterations of urinary secretions, polyuria, frequency of micturition occur. In the genital system we find impotence and ejaculatio præcox, and dyspareunia and vaginismus in women. In the limbs we find weakness, "giving way," paralysis. A feeling of general exhaustion is usual in depressive states.

A glance at this list will show that every region of the body may be the seat of neurotic symptoms, and it is true that any symptom of which a patient may complain may depend on his moods and not on any physical agency. It behoves us therefore to be on guard.

These responses to emotion may be comprised under the name emotional reaction. Every one of us is liable to some of them, but for most of us their duration is short. For many a flutter at the heart is common when called on to speak in public. But if we get up and speak, the symptom usually disappears. Just before an examination many candidates experience a desire to empty the bladder. For a large number the prospect of being shamed may

of those left have been braced by the discipline and hard life which broke the others. Some of course will be discontented, but most of them will survive at least to enter the danger zones. Some will break down sooner than others, and the factors which are of importance here are complex.

Leadership is one of these. There are leaders who inspire confidence and others who do not. It is safe to say that where leadership is good the percentage of breakdowns will be smaller than where it is poor. This question of leadership runs from the subaltern right up to the high command. The subaltern who knows his men well, who gains their respect and affection, will have few cases of breakdown in his unit. But it is necessary also that the unseen powers should make themselves felt. The strategists, the staff who work out plans, the commissariat, the medical organisation have obvious parts to play in the preservation of morale. Lack of confidence that these personages are playing their part will filter down to the rank and file, and in that event increase of neuroses may be expected.

By his very training we have caused the soldier to regress to some extent. By the word regression is meant a mental, either intellectual or emotional, moving back to childhood. The child is dependent, looks for guidance and protection, and as he moves towards adult life he should, if the process is smooth, become less dependent, more ready to make his own decisions and to assume responsibility. The fully fledged adult will seek advice only on purely technical points, e.g., whether or not he has appendicitis or what is the legal interpretation of some contract. On the important decisions in life he will weigh the evidence and settle for himself what he should do. Needless to say there is no such person—none of us arrives at the full adult position. But the soldier is asked to step back. He is to get up when he is told to. He is to go where he is told. He is to expose himself to danger or to refrain from doing so to kill or not as ordered. He is to make no decisions, only to carry out certain movements when the command is given. That is to say he is to regress towards childhood. There is one condition when it is easy to get children to obey implicitly and that is when they are quite sure that the parents will give them protection to the last drop of parental blood. The child does not reason this out, but the condition is there. When the child threatens some objectionable person that he will tell his mother

from that of the child for the former to have an inkling of what is going on in the child's mind. The only people who can bring up children are other children, and the function of the adult is to see fair play as far as possible. An average woman might bring up a real family, but one child is not a family, only a fractional family.

It is therefore difficult in such a case to say how much of the emotional instability of the child is inherited and how much acquired. It is of importance to realise that the acquired part is certainly there, for that is the only part we can deal with. I have noticed that those who lay stress on the inherited part and proclaim that it is the most important part, are also unduly pessimistic about the possibility of doing much by psychotherapy. Therefore the life history of any nervous patient is far more important for treatment than the family history, and neurotic parents are more important as sources of neurotic infection in early childhood than as progenitors. This is not to say that there are not bad stocks. It is only to emphasise that that is not the whole story, and that people with bad inheritance can still be treated successfully.

(2) The amount of emotional stimulus may be so excessive that even a person of good psychical constitution may be overwhelmed by it. In warfare the poor stuff will be weeded out early, some of it will disappear long before contact with danger is made. The voice of the sergeant, being expected to stand discomfort, cold and fatigue, the absence of affection—these things will bring the people we have just been considering into such trouble that they will soon be removed from the effectives. From every point of view it would have been well if they had never been enlisted. Some are difficult to detect, but there are a number of quite obvious misfits who would have been rejected were not examining boards so exclusively concerned with certain standards of physical fitness. Not only are these people a burden to the army, but also to the country. Many of them were before enlistment sufficiently stabilised to do useful work if left to do it quietly. That stability, such as it is, will disappear when they have first been punished for petty infractions of discipline and then recognised as mentally unfit for military life, and it will probably take a long time before their former stability is restored—if it ever is. I do not say anything about the misfortune for the individual himself. That is obvious.

After these unfortunates have gone, it will be found that many

some cases physical lesions were produced in the encephalon or spinal cord from a blast which had not broken the skin, it was at first assumed that a physical lesion had occurred in all cases. A study of "railway spine" a neurosis following railway accidents, might have prevented this mistake. The parallel between railway spine and shell-shock is clear. In both there is a history of an accident, and after the first shock has passed off there is no sign of physical injury. In both instances the doctors are puzzled and their explanations range from malingering to cerebro-spinal damage. In both the patient has something to gain by being ill, in one case money in the other escape from an intolerable situation. In both there has been a long series of bad suggestion, direct and indirect. In both many recover when the anxieties have been relieved.

It therefore behoves every doctor to take care of what he says to people after a serious accident. His slightest suggestion that the patient is worse than he thinks he is may be followed by far reaching results. It is his duty, if he is sure after a day or two that no damage has been done, to tell this to the patient in the most positive way and to emphasise that his recovery will be rapid, permanent and complete. It is significant that where there is no compensation of any kind to be obtained remote symptoms do not occur. They do not occur after skiing or hunting accidents. It looks then as if the possible exploitation for gain were the important factor but this would not bring about the symptoms if the proper suggestions were made at the beginning, if the desire for gain was not aroused by the doctor or the solicitor.

From a very large number of histories I was able to convince myself that the notion, still to be found in the psychological writings of certain authors who were certainly never in a position to examine these patients, that the men, who became ill after being blown up were not consciously afraid until this had happened, may be dismissed as a myth. I have seen it asserted in more than one place that these patients had been suffering from unconscious fear before their accident, and that in some mysterious way this fear had escaped from its repression into consciousness as a result of the catastrophe. This view may be regarded as an example of wishful thinking on the part of those who cannot believe that a neurosis can be precipitated unless there be unconscious factors. I believe this to be wholly untrue for two reasons first because,

he is showing this complete reliance on the parent's being both able and willing to protect. Every officer in the army is a parent surrogate for every person under him. The keen-witted French seem to have recognised this when officers call their men *mes enfants*.

There are many physical things that the good parent substitute will see that his children are provided with as far as is possible. Some of these have been mentioned, good food—good in every sense of the word, properly cooked, of appetising quality, sufficient in quantity, and with as much as possible of the accessories to the calories about which we have learned since the last war. When the children fall ill he will see that they get proper medical attention. Since the last war I should have hoped that this would include proper psychiatric attention, for there seems to be no doubt that the Americans, who had profited by our mistakes, had a smaller incidence of severe neurosis, because men were encouraged to report their mental difficulties, their insomnia and so forth, and to discuss these things with the psychiatrists.

(3) Prolonged ill health of subacute character does tend to produce nervous symptoms. Acute ill health like pneumonia of course does so also, but in such cases the major condition dominates the picture, and there is no likelihood of the condition being overlooked. Men, however, who have had malaria or dysentery or trench fever, whose acute condition is past, may yet be far from well, and these patients may easily become the prey of neurosis superadded to their physical state. Hardship and misery are less easily faced by those whose health is even a little subnormal.

(4) Shattering experiences, being blown into the air or buried by a collapsing trench, were in the last war almost universal features in the history of the soldier with a nervous breakdown. So constant was this story that, as is well known, the term "shell-shock" was for a time universally used as a synonym for war neurosis. A man was supposed to be well and happy, a shell burst near him, and thereafter he was ill with a very intractable set of symptoms. The evil effects which followed such an experience became more widespread as the war continued, until the authorities were compelled to introduce a definition of shell-shock, which you were not allowed to have unless you were blown so many feet away from where you had been standing or been buried for so many minutes.

The neurologists were somewhat to blame for this. Because in

were not safe, as was shown clearly by the marked exacerbation of anxiety and its bodily accompaniments during an air raid.

The physical symptoms were protean and were general and local. A general one of great importance was exhaustion, which will be dealt with in detail later.

Connected with the head the most prominent were headache, failure of concentration, forgetfulness, insomnia, terrifying dreams. These symptoms led on to other anxieties, that some damage must have been done to the brain which would lead to mental deterioration or to insanity. It is not surprising that a man who feels that his brain is not working smoothly should fear he is becoming insane. The laity think that a man can know when he is doing so and that no brain could possibly stand what his is being asked to put up with. This fear increases the anxiety and this added anxiety makes him more sure that he will become insane, and so a vicious circle is set up which can go on indefinitely. The fear of madness leads in its turn to fresh anxieties. Almost every uninstructed person believes that a person may go mad suddenly, and unexpectedly commit suicide or make a murderous assault. That they will do so provides an added terror for these patients. Thus we can see that the symptoms of nervous untreated tend to get worse and worse. It is like an avalanche, beginning with a small movement of snow and adding more and more to the speed and quantity of the moving masses till the whole hillside is engulfed. And yet it is easy to break up a complex of this kind if anyone knows it is present. Patients, however, will seldom volunteer such a story. They believe that they are going mad and that the less they say about it the better, that if they tell the doctor his suspicions may be aroused and before they know where they are they will be whisked off to an asylum. Therefore it is better to say nothing. I have never seen a patient suffering from failure of concentration and insomnia who did not have or had not had this fear of insanity and though they seldom spoke of it spontaneously there was no difficulty in eliciting the fear by direct questioning.

The insomnia was seldom absolute. The patient would fall asleep to be wakened soon out of a terrifying dream. These dreams were often, as has been said already, of a peculiarly loathsome character. The man would be in a trench full of corpses from which rats would be emerging. He would be the only living person there. Shells would be bursting around. Dead friends would grin

having taken personally the history of some hundreds of these soldiers, I never failed to find a prolonged history of conscious fear and terror in existence for weeks before the accident, except in men who were resistant and would not talk at all, secondly, because treatment which did not concern itself with the unconscious resulted in large numbers becoming well.

The histories of these men were sometimes difficult to get. Many of them were trying to "put the war out of their minds," because they had been told to do so—a thing easy to say but by no means easy to do. Others were afraid that the doctor was going to suggest to them that they were cowards, a thing most of them had been fearing, a subject which they were terrified to discuss. They would refuse to talk about the war, would say they were not interested in it, that they were out of it and preferred to think of other things. It is curious to observe how often neurotic people say that they are not interested in something, when the truth is that they are averse from discussing it, often from fear, often from some other inhibiting emotion such as shame. It was easy to show these men that they were in fact deeply interested in the war. Most of them dreamt of it every night in the most vivid way. There was no need to make any interpretation of these dreams. The men were quite openly back in the trenches, in the mud and filth with shells bursting around, in a horror which so exceeded reality that they wakened shaking with fear, leaping out of their beds, hiding beneath them, and showing every symptom that the war was on their minds very much indeed. When one had got so far it was usually easy to persuade them that the plan of trying to put the war out of their minds had failed, and that it might be of more value to discuss exactly why they were being so overwhelmed by anxiety about it. This commonly resolved itself under a very few heads. They were full of self-reproach for having failed. They feared they had been cowards. They dreaded having to go back. They feared very much that they would be sent back before they were well.

### Symptoms of the Anxiety State in War Neuroses.

In many cases conscious anxiety was present, and, whatever its ultimate analysis, it was connected with a perfectly rational fear—that so long as the war continued there was a liability that they might be overtaken by it no matter where they were. Coupled with this was a fear that contempt might be shown towards them if other people knew of this. Even in hospital in England they

that it must be emphasised that as a rule what may be called a *negative reassurance* to a neurotic person does not have the slightest effect on a symptom. There were no doubt many soldiers who had had a physical cardiopathy, which recovered, and the symptoms disappeared. We are now considering however people who had that amount of anxiety which leads to neurosis. Their anxieties referred to two things—going back to the war and the heart itself. The first anxiety may or may not have been in consciousness. In most cases it probably was not. The anxiety about the heart itself would probably fill consciousness so fully that there was no room for any other anxiety. Let us think of such a man. The slightest exertion gives him violent palpitation and shortness of breath. When he walks about he is often giddy and has to clutch at the furniture. Sometimes, even when sitting quietly his heart begins to thump “turn over” and behave in other distressing ways. He gets a pain below the left breast, and he has heard of that fatal condition *angina pectoris*. And yet these doctors tell him that his heart is now normal. How could he be expected to believe them, be they the most famous cardiologists in the world? Yet in many the most elementary psychological explanation will cause all these symptoms to disappear.

After a rapport has been established—how this is done will be explained later—the patient is reminded of what of course he knew quite well, that emotional states like fear will cause palpitation of the heart, and that there is no doubt that he has been very frightened indeed about his heart. It must be explained to him that he was in the circumstances perfectly justified in being frightened, but that it is quite certain, seeing that these cardiologists are sure that the heart is all right, that the present symptoms are caused by these fears, that when he walked up a few steps the symptoms came on, not because he was straining his weak heart, but because he feared he might be. The psychotherapist need not examine the heart itself, in fact he had better not. He explains that that has been done by heart experts much more expert in this subject than he is, men whom he trusts absolutely men whose error might lie in the direction of finding heart disease when it is not there rather than in missing a real physical cause.

Some patients will accept this straight away others will try to defend their symptom because they do not want to lose it. They will say “Very good, but all this palpitation I have had, all this

at him Quite often these dreams were undistorted experiences which the man had gone through, thus differing from the dreams of adults in peace-time where, though events which have happened in waking life are dreamt about, these events are always distorted and jumbled up with other events Children normally often have undistorted dreams, and the fact that these soldiers did so affords some ancillary proof of that regression to childhood which military training induces.

There were also in some soldiers' dreams examples of symbolic representations of the patient's anxieties, which with a little talk were easily understood. Many men dreamed that they were back in the trenches in the blue uniform of the convalescent Without help from the doctor many recognised, when asked of what the dream made them think, that it represented the almost universal fear that they would be returned to duty before they were well Other men dreamt that they were in civilian clothes, which usually, but not always, yielded the interpretation that they hoped that the war was over and that they were back in civil life. I had at least two patients, who said that they wished they were in civil life and that the war had not yet started but was about to start Both were men of what is called the public school type Both were privates after a year or two of service, and all their old friends were officers They wanted a second chance, in which they did not doubt they would do better There were several patients who dreamt that they were in a mixed clothing of uniform and "civies" They would be in complete "civies" except perhaps for one puttee or an army cap All these men had seriously contemplated desertion But you can't really do it There will be something that will give you away

The effort syndrome was common and not always easy to diagnose The various mild infections from which so many men had suffered made it possible that there was a physical interference with the heart's functions. However important this may have been at the outset, it is certain that later the symptoms were frequently kept in being by psychogenic factors. The man may have had something wrong physically, which had necessitated care, rest and the absence of over-exertion. It became fixed in his mind that he had a weak heart, and no amount of explanation that it was now well did in fact make any difference The reason why it did not was in most cases a little complicated, but before going into

that it must be emphasised that as a rule what may be called a *negative reassurance* to a neurotic person does not have the slightest effect on a symptom. There were no doubt many soldiers who had had a physical cardiopathy which recovered, and the symptoms disappeared. We are now considering, however, people who had that amount of anxiety which leads to neurosis. Their anxieties referred to two things—going back to the war and the heart itself. The first anxiety may or may not have been in consciousness. In most cases it probably was not. The anxiety about the heart itself would probably fill consciousness so fully that there was no room for any other anxiety. Let us think of such a man. The slightest exertion gives him violent palpitation and shortness of breath. When he walks about he is often giddy and has to clutch at the furniture. Sometimes, even when sitting quietly his heart begins to thump 'turn over' and behave in other distressing ways. He gets a pain below the left breast, and he has heard of that fatal condition *angina pectoris*. And yet these doctors tell him that his heart is now normal. How could he be expected to believe them, be they the most famous cardiologists in the world? Yet in many the most elementary psychological explanation will cause all these symptoms to disappear.

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Some patients will accept this straight away others will try to defend their symptom because they do not want to lose it. They will say "Very good, but all this palpitation I have had, all this

overwork my heart has been doing will surely have strained it" That must be met with denials. Palpitation of this kind may go on for years, and when it stops the heart is all right. The patient may tempt the doctor to re-examine the heart. This should be refused absolutely, and the reasons already given should be repeated. If the doctor yields and examines the heart again, then the patient will know at once that he has not wholly accepted his cardiac experts, and the reign of doubt and anxiety will begin again. This is a universal rule in psychotherapy. Examine if you like, have as many experts as you like, before giving the opinion. But once the diagnosis of neurosis has been delivered to the patient, do not examine any more. All you will do is make the patient conclude that you are not sure.

It is no violent assertion to say that the patient has difficulty in believing that he has no physical illness because he does not wish to lose some advantage which the illness is giving him. It is sometimes said that he does not wish to be well, a statement which may be described as nonsensical and one which always makes a patient more difficult to deal with. Everyone wants to be well, but everybody is not prepared to accept in all circumstances the consequences of being well. No one enjoys cardiac discomfort, but if weighed against the hell of front-line trenches, it may be the lesser of two evils. If the explanations about the heart have failed to produce amelioration, it may be pointed out that there must be some other anxiety which is keeping the symptoms in being. If the patient says there is none, the direct question may be put as to whether he is not anxious about the thought of returning to duty. Further discussion on this hand will come in the paragraphs dealing with treatment of war anxiety in general.

An example may be given illustrative of these points. A man was admitted to hospital for some trivial condition and knew he would be returned to duty in a few days. He was appalled at the thought. In hospital he was told that if one inhaled strong pipe tobacco, one was pretty sure of acquiring a cardiac disorder. The experiment was entirely successful, and he kept it up long enough to ensure his return to England. Here then we start with a physical poison. After a time he stopped inhaling, but the heart did not improve. In due course he was told the heart was normal, but as the symptoms continued he was invalided out. Several physicians of standing told him his heart was normal and his symptoms

disappeared. The war terror, however remained. He thought he might be re-enlisted and he managed to find a very young doctor with no cardiological experience to say that a cardiac disorder which had lasted many months might mean that there was something wrong with the heart and that the symptoms might return. They did so in a few days and persisted for the duration of the war. By that time they were fixed, and the end of the war did not see the end of the symptoms. He had now become sure that by his own act he had permanently damaged his heart. Shame and the fear of prosecution made him keep his guilty secret to himself for a year or two but before he became well he had to disgorge the whole painful story.

What has been said here of the circulatory system is equally true of all the other systems of the body. There were many physical causes for dyspepsia, but there was also a purely psychogenic dyspepsia. We know that emotion may affect gastric secretion and motility. Psychogenic dyspepsia is common. It is often psychogenic from the outset, but a physical dyspepsia may easily be the starting point of a psychogenic one. As an example of the first may be cited the case of a soldier in the retreat from Antwerp who drank water from a public tap as did most of his companions. When he had finished his eye caught a notice that this water was not fit for drinking. He was seized with pain and vomiting, but there were no others affected in this way as there would have been if the water had really been the cause of his attack. He was taken prisoner and in Germany he had prolonged dyspepsia due, he believed, to his stomach having been damaged by this poisoned water. He was unable to take enough food to keep himself even reasonably fit, and he became so ill and emaciated that he was transferred as an incurable to Switzerland, where he soon became quite well. He had, however learned the lesson that illness may confer advantage. It is not suggested or believed that he thought this consciously but that there is a tendency to repeat anything which conveys advantage even when it is not consciously recognised. When he returned to civil life after the war he fell ill with dyspepsia and loss of appetite at several crises in his life.

*Exhaustion*—This word is used in two senses. There is an objective exhaustion, as when an electric battery is run down or a mill-dam is empty of water. The potential energy has been used up and there is none left. There is also a subjective sensation of

being tired, and if people feel very tired they are apt to say that they are exhausted. But it must be seldom indeed that this is true. There must be few occasions on which further exertion could not be made if it were essential. In the great retreats of history men have marched night and day for long periods in a way that they could not have done without some special stimulus, which shows that much of the feeling of exhaustion in every-day life is not that potential energy has disappeared as it has in the example of the empty mill-dam.

The sensation of exhaustion may arise from many causes. All the fevers cause it. There are other physical morbid states which are accompanied by it. What is to be said now has no reference to these conditions. It is likely that they do not represent an utter emptying of potential energy, but they are physical inhibitions on the use of energy and are therefore to be treated with respect.

The sensation of exhaustion may also occur when the physical condition is perfect. It is common when we are worried, annoyed or bored, and in subjects who are easily alarmed at symptoms of any kind the sensation of exhaustion may be very great indeed. If such people fear that they may harm themselves by doing too much in what they believe is their exhausted, run-down state, they will feel exhausted very quickly as soon as they are asked to do anything extra, and now we are in the presence of a psychical inhibition of energy which need not be respected. This does not mean that the patients are to be told to get on with it, to pull themselves together. Obviously that must make them worse. They will try, but they will try in fear. Fear will usually in a neurotic overcome will and the symptom will increase the more the patient tries. An explanation will, however, usually remove the symptom unless the patient has a use for the symptom, such as keeping out of a war of which he has become terrified or receiving some compensation after an accident.

Two war examples will be given of chronic so-called nervous exhaustion, one beginning with an infective state which was allowed to drift into neurosis, the other after what was really over-exertion.

The first was a lad of twenty who enlisted in the first rush in 1914. He was given leave at Christmas and went home. Almost immediately he got influenza, and the day before he was due to return, as his temperature was still up, his doctor said he was unfit

to travel Word was sent to the authorities. In accordance with routine the local police were asked to call to ascertain the facts. He heard that this had happened and thought that he was being considered a deserter as he did not know that it was the usual routine. He therefore got up and returned to his camp feeling during the journey that his fellow passengers were eyeing him and seeing what a contemptible person he was. At the camp no notice was taken of him, and he paraded next morning as usual. He fainted on parade. I saw him eighteen months later when he was in a state of exhaustion with depression which had lasted during the whole period. He had done no soldiering during this time and was depressed. The story as told above had been accepted without further questioning, and the condition had been called weakness—though the word used was debility after influenza, and the case had been taken as additional proof of what everybody knew already that if you get up too soon during influenza the after consequences may be very serious.

A little investigation into the facts of the story showed that such an explanation of the case presented grave difficulties. When the patient collapsed on parade he was told to go and lie down on his bed. Apparently no one took any notice of the matter and, driven by his conflicting emotions, he did in fact not obey this order but went for a ten mile walk alone which makes it hard to believe that the collapse was due to any kind of toxic weakness. When he got back without having been discovered, he realised that this walk was an act of insubordination, which of course produced more physical reaction. This took the form of the feeling of exhaustion. For some time it was steadily pumped into him that the symptom was due entirely to getting up in the middle of influenza, and it was pointed out that people had to pay for this by a very long illness. Everyone during many subsequent months seems to have accepted this, till one day it occurred to a medical board that the patient was suffering from neurosis, seeing that depression had become so marked a feature in the symptomatology. It was now easy to get him to see that the influenza theory was just nonsense though as a theory but not as a fact it had made him ill, and that the illness had been started by fear of disgrace, and had been kept up by the fear that he had ruined his health for life. He recovered quickly.

The second case was that of a man who had become exhausted

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extent shall we pursue our punishing methods? I once had an interesting conversation with a rather truculent medical general, who hated neurotic patients, and was fully convinced that they should be made to pull themselves together and get on with the job. He asked oratorically what was to be done with them if they refused. I suggested they might shoot the lot. This, however was too much for the general, who said that the public would never stand it. In theory he seemed to approve.

### Diagnosis

The diagnosis of anxiety neurosis does not depend on our finding a patient ill without physical signs. It may be that we have not detected signs. It may be that they will be manifested later. Further neurosis may be the most important part of a case where physical signs are present. If, however in the absence of abnormal physical signs we get a bizarre story an over rich symptomatology a set of symptoms which make no kind of sense then we may suspect neurosis. There will also be the fact that the man is nervous, a thing he may try to conceal if the doctor is unsympathetic but if the doctor gains his confidence by showing interest, he will be rewarded by hearing how frightened the man has been for a long time.

### Treatment of the Anxiety State

The treatment of the anxiety state consists in finding out what the anxiety is about. The patient has a conscious anxiety that he will not get well, or that although he has repeatedly been assured that he will get well he does not know and no one seems able to tell him, how to do so. There may be no reluctance to get well, conscious or unconscious. It is simply that he does not know how to do it. On the other hand, there may be a conscious knowledge that the consequences of getting well may be disagreeable or there may be an unconscious desire not to get well. The first set of patients, those with no inhibition to getting well, are easy to cure. The second set may be extremely difficult. We shall not be able from the nature of the symptoms nor from their severity to gauge to which category the patient belongs. We shall discover this when we have discovered whether the patient accepts our explanations easily or not.

The treatment of the anxiety states differs from that of physical illnesses in that it goes on all the time we are making the examination of the patient. We cannot even examine this kind of patient

during the retreat from Mons ; the exhaustion had lasted for over three years. If he walked a couple of hundred yards he was utterly done up for the rest of the day. Cases of this kind were not uncommon. All sorts of theories were devised to account for them. It was held by some that those mechanisms within us, by means of which food is transformed into potential energy capable of being stored, had become overstrained by the immense over-exertion of an experience like the retreat, and that they had never recovered. The dynamo had been damaged, and now generated so little current that what it did was soon used up. Or it was supposed that these people leaked energy, the accumulator would no longer hold the energy, just like a battery with faulty plates. It never seemed to occur to these theorists that the cardiac and other vital processes went on as usual. There seemed to be plenty of energy for them.

Everyone was exhausted after prolonged over-exertion, but the majority recovered in a day or two. When men such as the one whose case we have been considering were told that they remained exhausted because they feared that they might do themselves great damage if they did too much, and that the sensation of fatigue came to them as a defence against damaging themselves, and if this information was coupled with the idea that it was unlikely that they would be sent back to duty even if they did get well after so long a period of illness, they usually did get well, as in fact this man did.

It may be felt by many that a man like this was a malingerer, and that he got well because he was almost promised that he would be discharged from the army if he did. There is a theoretical distinction between malingerer and neurosis, namely that the malingerer is fully conscious of what he is doing and the neurotic is not. Some say that we have only got the man's word for it. In some cases that is true, but in others it cannot be. No one could keep up voluntarily the coarse tremors all day and every day as some of these anxiety patients did. So it is clear that there are some cases of anxiety neurosis in which we have something more than the patient's word for it. Even if we never had more, we have only shifted our difficulty. As soon as we have arrived at the diagnosis of malingerer, we are of course able to relieve our own feelings by much righteous indignation. We can feel that we are justified in punishing the man. But when we have done this shall we make either a good soldier or a good citizen ? And to what

required the shattering experience of being blown up to make them feel very ill and to make them think they could now go back to England honourably

From later experience it was generally agreed that these patients should not have been sent to England, that as soon as they were capable of understanding what was said to them, they should have been repeatedly assured that nothing serious had happened. They should have been induced to get about as quickly as possible. This, however was not done till towards the end of the war. The patients in the first years were evacuated to England everything on the journey was done to fix in their minds that they had been seriously injured. If it had been generally understood that "shell shock" was not a method of getting to England, there would have been much less of it.

If a man has had it emphasised both by word and deed that he is physically ill, it requires something more than a contradiction by another person to make him change his mind. The prophylaxis of the war neuroses therefore becomes a point of importance.

The battalion medical officer should try to recognise when a soldier is showing signs of nervous wear (Blacker 1943). During the last war the battalion medical inspection was apt to be conducted on the lines that if a man had no physical signs he must go on with duty that skrimshankers were to be discouraged. It may be difficult but it is not impossible, to distinguish between the men who would like a few days off duty and the men who are really beginning to break down. It is quite likely that the men who are on the verge of a breakdown will not report themselves sick. They are too afraid or ashamed to do so. The medical officer if he gets to know his man properly should certainly spot some of them, and the company officers should be able to help him to get on their track. This would require on the part of everybody concerned a re-orientation of ideas about neuroses. It should no longer be considered disgraceful to have neuroses. Neurosis is a condition requiring treatment. The man should be interviewed, allowed to say his say possibly be given a sedative. If he has been for a long time without sleep it should be arranged that he can have sleep. The mere fact that he can talk to someone about his fears and feelings will in many cases help to abolish them. In psychotherapy it is just as important that the patient should talk as that the doctor should. The patient discharges by talking some of the perilous stuff that weighs upon

until we have put his mind to rest in some measure, and as soon as we have put his mind to rest in any direction we have begun a certain measure of treatment. The reader will have noticed that in the description of symptoms, it was impossible to keep out considerations of treatment. Much of treatment has therefore already been given.

How are we to put the patient's mind to rest so that he will talk? We are to do so by making him feel that we are interested in his case and that we want to hear his history. In civil life this may take a long time, but in the war this was not common. The essentially neurotic people, the people who would have broken down with the strains of ordinary life, have probably been eliminated at the outset, and the patients left will in the majority of instances be people who developed neurosis only because of the enormous stress of war. Even so most of them need not have been so ill, need not have been so resistant to treatment if their cases had been managed differently from the beginning of the illness.

We may begin with the word "shell-shock," that unfortunate word, as has been already said, which made all people who heard it—and that was everybody—consider it a thing which must necessarily entail a long illness, and which would assuredly get one sent back to England. All this produced a devastating mixture of fear, hope and shame. These emotions were all in conflict, and therefore much anxiety ensued.

After a time most normal men wished that the war was over. Most men were reconciled to a disablement, if it did not give too much pain or was not of an appalling nature, if it ensured their future absence from the front. I do not think that blind men became reconciled to blindness, though there is a myth that the blind are cheerful. But most normal men did not mind a "Blighty one." There was therefore in most a hope of getting a not too serious wound or illness. There was, however, fear that one might not get sent home. Both these emotional states were to some extent general. The hyper-conscientious, the people who think that an unworthy thought is as bad as an unworthy deed, added shame to these two. Most people know that they will have unworthy thoughts, and are too sensible to bother about them. These others consider the thought nearly as bad as the deed, and are apt to label themselves cowards. Shame is a very devastating emotion, and all these people were very ready for a breakdown. It only

required the shattering experience of being blown up to make them feel very ill and to make them think they could now go back to England honourably

From later experience it was generally agreed that these patients should not have been sent to England, that as soon as they were capable of understanding what was said to them they should have been repeatedly assured that nothing serious had happened. They should have been induced to get about as quickly as possible. This, however was not done till towards the end of the war. The patients in the first years were evacuated to England everything on the journey was done to fix in their minds that they had been seriously injured. If it had been generally understood that "shell shock" was not a method of getting to England, there would have been much less of it.

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the heart " If this fails he should be sent to a hospital for functional nervous diseases, which ought to be near the front But he should understand that people will not be sent to the base on account of neurosis, that they can be cured at the front, and it should be made clear that in no circumstances will there be a disability pension for neurosis.

The last clause will require a re-orientation of thought on the part of a good many people besides the soldiers, and no class of people require re-education on this point so much as the lawyers For many years now they have been laying down that neurosis is as much a disability as a physical injury is, and therefore that it should be compensated in the same way. The premiss is true, the conclusion is false, there is no such consequence to be deduced that because A is as real as B, therefore their treatment should be the same. Somehow the lawyers must be taught that the more you pet and coddle neurotic patients, the more neurosis you will have This does not mean that the treatment should be harsh, it only means that it should be explanatory. We doctors are of course at fault also There are doctors who have taught lawyers the doctrines that neurosis should be compensated

If patients are seen within a few hours of being blown up or buried, they will probably be in a state of shock They may or may not have had a period of unconsciousness from concussion As soon as they have recovered they will presumably be examined, and it must now be decided what is to be done if there is no evidence of a physical lesion From the outset they should be assured that they have not been damaged, and that they will be quite well in a day or two Patients with concussion should be kept in bed for about a week. Those who had shock only should be got up as soon as possible They should be told that later symptoms will not develop in their case The "railway spine" tradition that people may start curious symptoms later on is possibly more or less familiar to many of them, and it is therefore advisable to say that in cases like theirs this will not happen It should be made quite clear that they are not going to be evacuated, but that they will return to duty soon

The doctor must train his non-medical assistants, orderlies and nurses, that they are not to make remarks which can in any way suggest that some disability may follow A corollary from the "railway spine" episode is that in no circumstances should a

neuroses be pensionable. The politicians will probably see to it that this course is not followed, but the medical view should be recorded

It is not likely that a counsel of perfection like the one sketched out above will be generally adopted and it is likely that considerable numbers of neurotic patients will be sent to safe places. When they have got there, the position to be faced by the doctors is a wholly different one. If the patient is suffering from much obvious anxiety it is not likely that he will again become returnable to military duty. As has been indicated the syndrome will have become fixed by the time the patient gets to the safe place. Partly because of diagnostic difficulties it is likely that he will have been to several ordinary hospitals before he gets to a place where psychotherapy is practised. He will have had a number of physical diagnoses, from which he can with justice draw one obvious conclusion, that his case is a great puzzle to the doctors, that none of them knows what is really wrong. It is, however, fairly certain that the method of approach recommended here will not have been tried, the method of asking him to tell his story in his own way, of encouraging him to speak out freely his grievances and fears. He must receive no criticism. When he says he would rather not talk about some subject such as the war it must be pointed out very gently how unsuccessful the attempt to put it out of his mind has been. It may even be said to him that he is probably thinking the doctor regards him as cowardly but that this is certainly not the case. When the story has been told it can be pointed out that he has been experiencing very violent emotions, and the information can then be given that all the symptoms of which he is complaining can be caused by emotion—that all these worrying diagnoses—worrying because all different—would help to keep the symptoms in being—and also by this time it might be possible to point out that there is the crowning worry of having perhaps to return to duty.

Slowly and reluctantly in the last war the military authorities came to the conclusion that it was useless to return these men with chronic neuroses to duty. A medical general came almost secretly to the hospital where I worked, and said would we please discharge as many from the army as possible, though we must not say he said so. There was at that time plenty of employment available, and when men saw that most of their fellows were being

discharged, they very easily accepted the view that their illness now depended on fears connected with the illness itself. From an early date I had assured myself that these men would not make satisfactory soldiers, and at the boards I always did my utmost to secure their discharge. I believe that such success as I attained in curing these men was due in part at least to this attitude which I took up, though before the visit of the general the matter was more difficult.

With regard to patients with gross hysteria, so long as they were paralysed or otherwise totally disabled, there was seldom any manifest anxiety. Some of these patients had no anxiety about returning to duty. These are dealt with in other chapters. There were, however, others in whom anxiety appeared very soon after the disability was removed by persuasion. I had several patients under care with paraplegia, who, while the condition was present, would say that they regretted being there in comfortable beds while their friends were toiling in the trenches. This was probably a genuine belief. When the paralysis was cured by persuasion they were delighted and remained so for a day or two. This shows that so far as consciousness was concerned they did not wish to be paralysed. Then they began to show symptoms of anxiety, which nearly always depended on the fact that they had come to realise that they were now eligible for duty. They began to have insomnia, to feel ill and so on. A neurosis is often a protection against something, in this case it was a protection against having to return to warfare, and so long as they were totally paralysed they did not need the protection of any kind of illness. Therefore anxiety did not manifest itself in consciousness so long as it was not needed.

When we have cleared up the anxieties of which these are samples we shall find the patient well. It is not necessary to tell him what to do, he will know and be willing to do it. There may, of course, be unconscious fears and anxieties, but I do not think the deep-seated ones are common in war cases. A soldier is on the whole a picked person. He is not likely to have been a born neurotic of the kind that produces the difficult cases of civil life. It is to be hoped that the medical officers who examine recruits will keep the latter out of the army.

POSTSCRIPT *January, 1941*

Since this chapter was written I have had the opportunity of seeing many officers who have developed neuroses during the present war. A considerable number had been sent straight from the ships after the retreat and evacuation of the Army from France. Many had been blown up. Many had had concussion, which had perforce been treated in the worst possible way as they had been obliged to march as soon as they recovered consciousness. When these men arrived they were down and out. They had not slept except in snatches for about three weeks. They had had little food. They had fought and marched the whole time. They were completely exhausted and only too thankful to drop into their beds. They did not want to talk, and it was arranged that they should lie down and sleep for two or three days. Some slept naturally at once. If they did not, they were kept asleep with hypnotics.

When they had slept off their exhaustion, they were examined and given reassurance of the kind already described. They were especially told that as they were uninjured no harm would come of what had happened. Their stories were listened to. They were very hostile to authority. They felt that they had been let down badly. The strategy had been ill conceived, the staff work bad. The generals had been overborne by the politicians. They should never have left their prepared positions in France for what they said were purely political purposes. This sort of talk was listened to without comment, and in a couple of weeks the sense of grievance had disappeared, and most of them were clamouring to get back to duty.

There were some who did not improve. Among these were elderly officers who had rejoined after having retired and were not up to the strain. Others had a history of breakdown in civil life, a history which had, unfortunately not been weighed when they applied for readmission to the Army. An officer of forty two had been in the last war, had continued in the Territorials for seven years afterwards and had then resigned but had remained on the reserve. A year before war broke out he had suffered from a nervous breakdown and had been away from his business for nine months. He had been back at work for only three months when he was called up. He never said anything about this breakdown, as he was anxious to do his bit. The retreat shattered him, and

he remained a person easily agitated with most of the signs of chronic fear. There were several officers with similar histories who did not stabilise sufficiently to justify retaining them in the Army.

With regard to the other officers who presently went back to duty, I could not escape the conclusion that, if they had been told anything else but that they had not been damaged, that there would be no sequelæ, and that they would get well, they would have passed into a state of chronic neurosis. Many had premonitory symptoms such as tremor, insomnia and unreality feelings, which might have proved to be the seeds of a lusty growth of chronic nervous disorder had they not received immediate reassurance that their symptoms had no sinister significance, that they did not indicate that they had received any injury to the brain or spinal cord, that their symptoms were only those of exhaustion and of having been subjected to a period of emotional strain of an extremely severe kind.

I cannot overstate the importance of emphasising these views to the patient as soon as he is fit to receive them. As we shall see in a moment and again later when we come to consider the civilian cases, the whole success of this treatment consists in giving it at once and giving it without any hint of doubt. As soon as the doctor is sure that there has been no injury capable of producing the symptoms presented, he must tell the patient that he will be well. There are some who do not like to tell patients that they will be quite well immediately or at longest in a week. Their minds are too apt to dwell on remote possibilities of untoward happenings. The doctor who hedges, who can never quite make up his mind, but always leaves a loophole, is a great creator of neurosis, and there is no place for him in the treatment of people who are only exhausted and who have just gone through terrifying experiences.

In contrast with these patients who were cured quickly, there were a few who were neglected at the outset, having been sent home instead of being sent to hospital. Nothing special was done for them. If there was any idea at all in the minds of those who sent them home, it consisted in a hope that time would work a cure. I suppose that sometimes this may happen, but if it does not, it usually makes things worse and does not merely leave things as they were. When these patients did come to hospital, not until two or three months after arriving in this country, their

symptoms were fixed and they presented all the difficulties with which we were faced in the last war

The Dunkirk men have now passed beyond my ken but I heard from several at Christmas 1940. Those who wrote were well and happy and though I have no statistics I am inclined to think that most of them have kept well.

I do not know anything about the rank and file as I have had no experience of them this time. But in this connection we may consider what has happened to the rank and file of the front-line civilians in the bombed cities. With rare exceptions they have not developed chronic neuroses. People in a state of panic have rushed to their local hospital, one of which I visited. Dazed people and unconscious people have been brought in. The same treatment of a short rest with sedatives, followed by intensive reassurance, has been given, and as quickly as possible they have been sent back to work. Here too the treatment has been successful. This is gratifying from more than one point of view. It is, of course, a good thing that these people have been saved from months of invalidism. It is encouraging that so many doctors have freed themselves from the shackles of an almost universal mechanistic pathology which did so much harm in the last war all the more as this pathology was largely a fanciful one. It is also a matter for admiration that courage should be so widespread among our people. Before the war it was thought that the great cities would be overwhelmed with panic-stricken mobs. It is clear that we knew little of the reserves of courage that were present.

During the last few months we have had officers in the hospital who are ill because of the war though not because of warfare. Their cases have resembled those found in civil life. They are exhausted, sleeping badly depressed. The causes vary. Many of them have not had enough to do. In some units the officers say that their days are very full indeed, but there seem to be others where the commanding officer is not a very energetic person, and his keener subordinates have found time heavy on their hands. Many of them have been greatly irritated by what they felt was wasted time. There is reason to think that this cause of neurosis is getting less.

There have been others who have complained that the work they are doing has little relation to their abilities. A building contractor in a large way a motor engineer in charge of a fleet of

lorries in peace-time, men of over forty have been sent to give recruits squad drill. A business man of forty-eight was made a lieutenant of engineers and put to learn mathematics. He was, of course, hopelessly beaten by the young lads of twenty and lost heart. These misfits among a large number are probably inevitable, but it should be noted that neurosis can be engendered in this way.

On the other hand, I have seen several officers who had been suffering from overwork. This is a rare cause of neurosis, but it can happen. There were over-conscientious regulars, who had been given tasks in connection with the rapid expansion of the Army. For eighteen months or more they had worked fifteen hours or more seven days a week till they broke down. Anxiety played its part here, too. They had to accumulate certain materials, and these were frequently not to be had. They were treated with rest and encouragement and most are progressing satisfactorily.

By breaking up family life war has also a serious effect on certain people. I have seen several who developed feelings of jealousy about their wives, sometimes justified, sometimes not. But a man prone to this easily becomes a candidate for neurosis during enforced separation. The treatment for all these must be on the same lines as obtain in the treatment of the neuroses in civil life, only we have to recognise that the break up of ordinary social routine may be fraught with special difficulties for certain people.

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## CHAPTER XVI

### DIGESTIVE DISORDERS IN SOLDIERS

In the war of 1914-18 gastric disorders were comparatively rare among soldiers. In contrast with this dyspepsia has been extremely common in the present war and the diagnosis treatment and disposal of the dyspeptic soldier have presented very difficult problems for solution. It appears to be equally common in the Royal Air Force and slightly less common in the Royal Navy. Its incidence is high both in men serving overseas and in those who have not been out of England. In some of the early convoys from France the proportion of digestive cases was as high as 40 per cent and the total up to the emergency evacuation of base hospitals in April, 1940 as 14.4 per cent. It was officially stated in May 1942, that 17 per cent of the total discharges for all diseases from the Army and the Royal Air Force and 13.8 per cent from the Royal Navy were on account of digestive disorders.

About half of the soldiers and sailors admitted into hospital for dyspepsia prove to have a gastric or duodenal ulcer although the proportion in published statistics has varied between 79 per cent. of 196 cases investigated by Newman and Payne early in 1940 and only 29 per cent of 217 cases seen in a special centre of an E.M.S. Hospital in 1941 by Morton Gill, Berridge and Arden Jones. According to Hartfall (1941) 60 per cent of dyspepsias in the Army are either purely psychogenic or have become psychogenic although primarily organic. This seems a much more likely estimate than the 16 per cent diagnosed by Morton Gill and his colleagues who on what appears to be quite insufficient clinical radiological and gastroscopic evidence diagnosed no less than 47 per cent as suffering from gastritis, gastro-duodenitis or duodenitis.

About 90 per cent of men admitted for gastric and duodenal ulcer had suffered from an ulcer before they joined the Army (Newman and Payne 1940 Graham and Kerr 1941) or Navy (Wade 1943) generally for several years, the average duration of symptoms being seven years. Most of them were reservists, who should never have been allowed to rejoin. Questions about previous dyspepsia had rarely been asked by medical boards. Many men did not like to complain of indigestion, and others did not mention

it as they were free from symptoms when examined. In some a gross error was made by the board in passing for service men who had had a gastro-jejunostomy or an operation for perforated ulcer (R. H. Wilcox, 1940, Graham and Kerr, 1941). As the higher age groups became conscripted for military service, the proportion of men suffering from ulcer increased, in spite of greater care being taken by medical boards to reject all men suffering from ulcer and all who could bring clear evidence of having had an ulcer in the past. Ulcers rarely develop for the first time in men after they have joined the Army, whether in peace or war. Colonel E. B. Marsh, for example, saw only two cases in British soldiers during his service in North-West India.

The cause of the recurrence of ulceration appears to be the change from the comparatively strict diet, which almost all the patients had followed for years in civil life, to the heavy army food. The quality of this was on the whole good, but the cooking was at one time almost invariably bad. Fortunately by the spring of 1941 it had greatly improved (Tidy). There was much complaint about the greasiness of the food, and the meat ration was excessive. Many men replace some of their rations by articles bought outside and so keep fit whilst they are still in England, but overseas this is generally impossible and a breakdown follows. There is often a considerable deficiency in vitamin C in the diet, fresh green vegetables and fruit being rarely provided, but it is not likely that this helps in the development of ulcer. Psychological factors appear to have been of little, if any, importance in the British Expeditionary Force in France, but this is certainly not true for many of the cases developing among soldiers now serving in England and North Africa. Recurrence of symptoms occurred rapidly after enlistment, in 70 per cent. within two months, and often within a few days, and in the remainder within eight months (Graham and Kerr).

### **Prophylaxis.**

No man who presents definite evidence of having had an ulcer should be accepted for service in the Services. It would be unsafe to rely on a man's own story for obtaining such evidence, as it is impossible to make a diagnosis with any degree of certainty from the history alone, and it would not be difficult for a man wishing to escape service to describe the symptoms of a relative or friend. Only if written evidence is obtainable from a man's own doctor

or from a hospital that he has had symptoms of ulcer and that the diagnosis has been confirmed by the x rays should he be rejected on this account. He should be rejected however long he has been free from symptoms, as the ulcer diathesis is always with him and makes him liable to recurrences, when the care he has been able to devote to his diet in civil life is replaced by the comparative hardships of military service whether in England or overseas. No doubt a small number of men who have had an ulcer may be unable to produce the necessary evidence but it would be impracticable to make a complete examination in every man who gives a history of dyspepsia. On the other hand, if a man states that he is actually suffering from symptoms suggestive of ulcer at the time of his examination especially if epigastric rigidity or well-localised tenderness is present a radiological examination should be carried out before coming to a conclusion. With these exceptions dyspepsia should not be regarded as a reason for rejecting a recruit.

#### Diagnosis and Treatment

When a soldier is admitted to hospital on account of dyspepsia, it is of the utmost importance that a definite diagnosis should be made with a minimum of delay. It should be possible with a well taken history combined with radiological examination, chemical and spectroscopic examination of the stools for occult blood, fractional test-meal and gastroscopy in doubtful cases if an expert is available, to decide with a high degree of accuracy within a week whether he has an ulcer or whether gastritis of sufficient severity to be the cause of his symptoms is present. Hartfall (1941) found that among 58 dyspeptics without radiological evidence of disease gastroscopy showed a normal mucous membrane in two-thirds, and less than half of the remainder showed a serious degree of inflammation. Other less expert gastroscopists have stated that hypertrophic gastritis is common in soldiers, whereas it is really very rare. This is almost certainly due to mistaking the thick but normal mucosa over a contracted segment of the stomach for hypertrophy although the thickening disappears on relaxation. The spasm is probably often the result of fright in anticipation of the examination. When gastritis and ulcer as well as cholecystitis and other possible organic causes of dyspepsia have been excluded, the dyspepsia can be regarded as functional, except in the comparatively rare cases of malingering.

There is a tendency in the Army to call all functional gastro

disorders "gastritis" But gastritis is an organic disease, as definite as ulcer, and it is most undesirable to confuse it with functional gastric disorders which have no organic basis. The same confusion was at one time common in connection with colitis, a term which should be reserved for actual inflammation of the colonic mucous membrane and which should not be used for the much more common functional disorders of the colon

When an ulcer is diagnosed, the soldier should as a rule be invalided from the service forthwith. There is nothing new about this, as it has been the general practice in the Army for many years. An exception may be made for officers, especially in the R A F, who are able to keep to a sufficiently rigorous regime to prevent a recurrence after a period of strict treatment in bed has resulted in complete healing of the ulcer. This applies also to men of other ranks, who can ill be spared on account of important duties of a sedentary nature and for whom arrangements can be made for the provision of well-cooked meals at regular hours with intermediate feeds. In the last war I saw several officers who had had a duodenal ulcer and who were able to look after themselves in France or in the East sufficiently well to remain free from symptoms. Graham and Kerr also noted that many officers were able to carry on in comfort in France in 1940 until severe physical strain and irregular meals in the weeks before the evacuation led to a recurrence of symptoms. A soldier with functional dyspepsia should be given a rapid course of treatment to restore his ability to eat ordinary army food and face ordinary army life. The malingerer, in the rare cases in which he is recognised with certainty, should be sent straight back to duty and the facts recorded on his army papers.

The investigation should preferably be carried out in a military hospital. There are to-day more expert gastro-enterologists in the Army than in civilian practice, and in many instances their expert knowledge is being wasted in routine and administrative duties. Moreover, there is no doubt that for the second group of cases—the functional dyspepsias—the discipline of a military hospital and treatment by an officer in uniform is far more likely to be effective than treatment in the very different atmosphere of a civilian hospital. My experience of visceral neuroses, both alimentary and circulatory, in the last war convinced me of this, and what I have seen and heard during the present war has only confirmed my conclusion. Delay in the proper treatment and disposal of func-

tional dyspepsias is likely to produce a soldier's stomach D.A.S. or digestion syndrome corresponding to so-called soldier's heart D.A.H. or effort syndrome" both are genuine enough, though they are badly named and the product of bad treatment and not of army life as such so ought never to be allowed to develop

I believe that an enormous saving of man power both for the Army and for civilian service with a corresponding saving in money and in occupied hospital beds would result from the establishment of special units, as suggested by Schindler early in 1940 staffed by medical officers with experience in gastro-enterology together with clinical pathologists, and expert radiologists, who should be capable of recognizing at least 95 per cent. of chronic gastric and duodenal ulcers. There should be three distinct divisions of the unit. The patient would be admitted into division A for diagnosis, where he would remain in bed for about a week on a strict diet but without drugs whilst the investigations were being carried out. When the diagnosis was established, he would at once be transferred to division B if suffering from organic disease and to division C if from functional dyspepsia. If he were malingering he would be discharged direct to his unit. A patient in division B would receive appropriate treatment, and if suffering from ulcer steps would be taken for his immediate discharge from the Army and his transfer to a civilian hospital where the treatment would be completed, after which he would be given a regime to enable him to keep well enough to do useful work in civil life. Patients with gastritis would generally be treated with sufficient success to make it possible for them to return in three or four weeks to full duty on ordinary diet. In division C the patient would be treated by medical officers with some knowledge both of gastro-enterology and simple psychotherapy. This should never last for more than three weeks, during the last of which he should be taught that he is quite capable of digesting ordinary army food in spite of any preconceived notions he might have to the contrary and he should be occupied all day. He would then be sent to duty with a note on his records that a full investigation had excluded organic disease. Only under very exceptional circumstances would he be sent back to hospital or reinvestigated in less than a year. A small proportion of cases admitted into division C might prove to be hopeless hypochondriacs, who should be discharged from the Army without a pension. The

value of such a special unit has already been demonstrated by Morton Gill, Berridge and Arden Jones in an E M S hospital (1942)

The United States War Department has approved a table of organization for general hospitals, which includes a Section of Gastro-Enterology in each 1000 bed institution. This section is to be directed by a major with a captain as assistant both of whom are "specialists in gastro-enterology or internists devoting particular attention to the specialty" \*

### CHRONIC GASTRITIS

#### Ætiology.

A man with good and sufficient teeth, who takes the trouble to use them and avoids grossly indigestible food and excess of alcohol, need not swallow anything until its thermal, chemical and mechanical properties have been so altered that it will cause no irritation when it comes into contact with the gastric mucous membrane. But many people bolt their food before it has been thoroughly masticated, whilst others have insufficient teeth and inadequate dentures, with the result that the stomach is daily subjected to mechanical, chemical and thermal irritants. It is also often damaged by excessive indulgence in alcohol, strong tea, mustard, pepper, curry, pickles, tough meat and uncooked or insufficiently cooked coarse vegetables. The stomach is frequently irritated by the unconscious swallowing of the "juice" of tobacco smoked in excess, especially when the stomach is empty, and by drugs taken, often quite needlessly, for supposed constipation, rheumatism and other self-diagnosed complaints.

The stomach attempts to protect itself by the secretion of mucus, and in the 80 per cent of people with an average type of stomach it generally proves successful. But the 10 per cent with constitutional hypochlorhydria and the 10 per cent with hyperchlorhydria are likely to develop gastritis. In most cases the hypochlorhydria sooner or later gives place to achlorhydria, and in gastritis associated with hyperchlorhydria the abnormally vulnerable mucous membrane is likely to be further irritated by the excessive acidity of the gastric juice.

Infected material is constantly swallowed by people with oral and naso-pharyngeal infection. In achlorhydria the antiseptic acid barrier of the stomach is lost, and streptococci from the mouth

\* *Amer J Dig Dis*, 1941, 8, 93

invade the stomach and irritant toxins are likely to form in the gastric contents. Acute gastritis, which is often followed by chronic gastritis, is a frequent result of acute food poisoning and of acute infections, especially influenza.

The causes of gastritis are so numerous and so common that few people can pass through life without being subjected to one or more of them. The 80 per cent. of individuals with the average normal stomach are so well protected that they are less liable to develop gastritis as a result of these insults, but few of those with constitutional hyperchlorhydria and constitutional hypochlorhydria escape.

### Symptoms

Chronic gastritis is often completely latent. When symptoms occur their nature depends upon whether the condition has developed in an individual with the hyperthemic gastric constitution—acid gastritis, or in one with the hyposthemic gastric constitution—achlorhydric gastritis. The diagnosis should be suspected from the history and can be sometimes confirmed by a test meal and radiology but proof can be obtained only by gastroscopy (*vide* p 177)

(a) *Acid gastritis*—Acid gastritis and duodenitis rarely give rise to symptoms unless they are complicated by acute or chronic ulcer. In the exceptional cases in which symptoms occur in the absence of a chronic ulcer they are indistinguishable from those of duodenal ulcer and a diagnosis can be made only when an x ray examination shows that there is no constant gastric or duodenal deformity in spite of the presence of typical symptoms tenderness and rigidity associated with hyperchlorhydria and sometimes occult blood in the stools. Haematemesis may occur from an acute ulcer or erosion, which gives no radiological signs of its presence but can be clearly demonstrated by gastroscopy but many hemorrhages of this kind prove on careful enquiry to be caused by aspirin (Hurst and Lantott 1939)

The treatment is that of ulcer but the period of bed and strict dieting need not be prolonged more than a week unless occult blood continues to be present in the stools. It is however essential that the patient should permanently follow the "post-ulcer regime" (p 189) as otherwise an ulcer will probably develop sooner or later

(b) *Achlorhydric gastritis*—The symptoms are mainly due to the achlorhydria and the complications to which it gives rise. Pain never occurs in uncomplicated cases, but slight epigastric discomfort,

generally described as fullness, pressure or heaviness, is common immediately after meals. It is often partially relieved by belching, but frequent attempts to eructate may result in aërophagy. In spite of the presence of achlorhydria heartburn and sour regurgitation, which are relieved by sodium bicarbonate, may occur. Nausea is not uncommon, especially in the early morning, and it may be the only symptom, but it is often associated with anorexia. Tenderness is slight and ill-defined, and there is no rigidity.

Constipation is generally present, but in one group of cases chronic or intermittent attacks of diarrhoea occur and may persist for many years with little or no gastric symptoms to suggest its gastrogenous origin.

In early cases a little free acid may be present in some of the fractions of a test-meal, but more often by the time the patient comes under observation achlorhydria is complete. In most cases mucus is present in the resting juice and in each fraction, and the curve of total acidity remains moderately high. In advanced cases, in which atrophy of the mucous membrane has occurred and involved the superficial mucus-secreting cells as well as the tubules, mucus is absent and the total acidity is much reduced. A histamine test-meal is unnecessary, as the injection never produces free acid when treatment with lavage fails, and treatment is occasionally followed by restoration of secretion in histamine-refractory achlorhydria.

### Treatment.

All possible causes of gastritis should be removed as far as possible. The teeth should be put into good condition and artificial ones supplied when necessary. Septic tonsils should be enucleated, and nasal infections treated. The food should be thoroughly chewed and eaten at regular times, the last meal at least two hours before going to bed. During the first week or two of treatment the patient should rest for half an hour after meals, and, if he is tired, for half an hour before meals.

The diet should be that of the post-ulcer regime (*vide* p. 189), but no drugs are required. In severe cases, especially if the stools contain occult blood, it is best to give the strict ulcer diet for the first week or two.

In achlorhydric gastritis with excessive mucus the stomach should be washed out every morning with dilute hydrogen peroxide, beginning with one drachm and increasing gradually to four drachms to

the pint. The test-meal should be repeated after the mucus has disappeared the secretion of acid returns in 80 per cent. of cases, often within a fortnight, but occasionally only after four to six weeks.

When treatment by lavage does not result in the return of secretion, and in all cases in which mucus is absent from the original test-meal hydrochloric acid should be given. A teaspoonful of dilute hydrochloric acid (B.P.) with a little sweet fruit juice, fresh when obtainable but otherwise bottled (in place of the sugar and orange-juice of peace-time) is added to about 5 oz. of water and taken as a beverage with the three chief meals.

### GASTRIC AND DUODENAL ULCER

#### Ætiology

A chronic gastric ulcer was found by Stewart in 22 per cent. and a chronic duodenal ulcer in 38 per cent. of 4 000 consecutive autopsies. The scars of healed ulcers were found in the stomach in 23 per cent. and in the duodenum in 3 per cent. of the series. It may therefore be assumed that about 10 per cent. of all individuals suffer at some time in their lives from a chronic gastric or duodenal ulcer. Duodenal ulcers occur between three and four times as frequently as gastric ulcers in better-class practice and between three and eight times as frequently in most British hospitals except in London, where for some unknown reason the incidence is about equal. The relative frequency of gastric and duodenal ulcers in soldiers and sailors approximates to that of provincial and Scottish hospitals, Newman and Payne (1940) giving a ratio of four duodenal to one gastric ulcer in 206. Graham and Kerr (1941) six to one in 158 and Saffley (1941) eight to one in 124 soldiers and Wade (1942) six to one in 471 sailors.

Though chronic ulcer is rarely diagnosed in children, the symptoms date from the age of 14 to 20 in about 12 per cent. of cases recognised later in life. The average age of onset of gastric ulcer in men is 45 and of duodenal ulcer is 38 but in 40 per cent. of soldiers it was under 25 (Newman and Payne).

Chronic ulcer frequently occurs in several members of a family in one or more generation (Hurst, 1921). Willcox obtained a family history of ulcer in 35 per cent. and of dyspepsia in an additional 40 per cent. of forty soldiers evacuated from France in contrast with 4 and 2 per cent. respectively among fifty soldiers with no

digestive disorders The ulcer is generally either gastric or duodenal in all the affected members, but a familial incidence is relatively much more common in duodenal than gastric ulcer (Nicol, 1941) In familial cases the symptoms tend to begin at an earlier age than in others, and there is a great tendency for anastomotic ulcers to form after operation

The peculiar type of ulcer found in the stomach and duodenal bulb occurs nowhere else except in the part of the jejunum immediately distal to the anastomosis after gastro-jejunostomy and partial gastrectomy, in the lower end of the œsophagus when relaxation of the cardiac sphincter allows gastric juice to regurgitate, and also in association with heterotopic gastric mucosa in Meckel's diverticulum The one common feature of these situations is the presence of acid gastric juice

Acute and chronic gastritis are frequently associated with localised loss of the superficial tissue, which varies in size from minute erosions only recognisable on microscopical examination to acute ulcers which are more or less easily recognisable with the naked eye The majority of erosions and acute ulcers heal rapidly, but if one occurs in an individual with the hypersthenic gastric diathesis it is likely to become chronic The various conditions already described which give rise to gastritis can thus also be regarded as factors in the development of chronic ulcers, especially of the stomach Among soldiers with ulcer the teeth were found to be inadequate for chewing in 38 per cent, an important factor in view of their heavy food and large meat ration Severe dental sepsis was present in only 13 per cent (Newman and Payne) In general, local irritation is the main exciting cause of gastric ulcer, whereas anxiety is of greater importance in duodenal ulcer The tendency to develop a chronic duodenal ulcer is increased by excessive smoking, the nicotine apparently acts through the autonomic nervous system by exaggerating the already excessive motor and secretory activity of the stomach This is a common factor in soldiers

### Symptoms of Gastric Ulcer.

The onset is generally insidious, the symptoms first appearing after big or indigestible meals The pain, which is often burning in character and may be very severe, is situated in the middle or slightly to the left of the epigastrium and may radiate upwards and to the back, it is much increased by indigestible food and

generally disappears with a milk diet. In ulcers situated near the cardia it begins almost immediately after meals, and in prepyloric ulcers two or three hours after, intermediate intervals indicating an ulcer on the lesser curvature. The pain generally disappears spontaneously after about an hour. It is completely relieved by vomiting and by alkalis, but as a rule only partially by food.

A small area of tenderness, the position of which is constant for each case, may be present in the epigastrium, especially in the presence of spontaneous pain. It is often associated with rigidity of the left rectus. When cutaneous hyperæsthesia is found, it has almost invariably been produced by the unconscious suggestion of the observer.

With increasing pain vomiting appears. It occurs at the height of the pain, a small quantity of acid fluid with a little well-digested food being brought up. When the pain is severe vomiting is often induced by the patient and may eventually develop into a voluntary act.

Occult blood is almost always found in the stools and disappears slowly when the patient is dieted. In about 25 per cent of civilian cases hæmatemesis occurs. When the blood is abundant it is bright in colour and the stools are tarry. When less abundant it is coffee-coloured and mixed with food. Melaena may also occur without hæmatemesis, but much less frequently than in duodenal ulcer.

The appetite is good at first and the tongue is clean, but fear of pain may lead to diminished intake of food with consequent loss of weight and weakness. In some cases, however, the relief given by food encourages the patient to eat heartily. Constipation is commonly present.

Conclusive evidence as to the size and position of a gastric ulcer is almost always obtainable by the discovery with the x-rays of a "niche" formed by the crater unless the examination has been postponed for a fortnight or more on account of hæmatemesis. In cases of doubt gastroscopy will settle the diagnosis.

A test-meal gives no constant result, but hyperchlorhydria is more common than in healthy individuals. In very chronic cases hypochlorhydria and even achlorhydria may be present, but these are caused by the associated chronic gastritis, as a second test-meal given after the ulcer has healed shows a considerable increase in acidity and often hyperchlorhydria, the treatment having led to disappearance of the gastritis.

### Symptoms of Duodenal Ulcer.

The earliest symptom of duodenal ulcer is generally a sense of discomfort or fullness three hours after the largest meals. This is gradually replaced by pain, which occurs between one and four hours after every meal, the interval being longer the larger the meal. It frequently wakes the patient in the early part of the night, particularly if the last meal is finished less than three hours before going to sleep. It is generally situated in the middle line rather nearer the umbilicus than the ensiform cartilage, it may radiate to the right or be situated on the right side only. The pain is often associated with a feeling of hunger and is relieved by taking food, it is therefore commonly known as "hunger pain". It is also relieved by alkalies and when the stomach is emptied by vomiting. Vomiting is common among soldiers (Newman and Payne), though rare in civil life in the absence of obstruction, and is often the determining factor in making a man go sick. It is mainly a result of unsuitable and excessive food, but in many cases it is in part hysterical (*vide p 193*), a combination of organic and functional conditions being present, as is so common under conditions of nervous strain. Constipation is almost always present. The appetite remains good, and the patient does not lose weight or strength.

Periods of hunger pain lasting some weeks or months alternate with periods of more or less complete freedom from symptoms. The attacks are more common in cold weather than in hot, but the autumn and early spring are generally more trying than winter. Attacks are liable to be brought on suddenly by worry, exposure to cold, acute naso-pharyngeal or bronchial infections, indigestible meals and excessive smoking or drinking.

In over 80 per cent of cases occult blood is found in the stools. Severe hæmorrhage occurs in 25 to 50 per cent of civilian cases admitted to hospital, but in less than 5 per cent of soldiers (Graham and Kerr). It always results in melæna and may also give rise to hæmatemesis.

Irregularity of the outline of the duodenal bulb, due partly to the deformity caused by the ulcer itself and partly to spasm, is always found. It affords conclusive evidence that an ulcer has been present, but unless it is tender or a definite niche is seen, the deformity may be produced by the scar of a healed ulcer. I have, for example, seen a grossly deformed duodenal bulb with no tender-

ness or niche in a man who had been free from symptoms since his ulcer healed under medical treatment thirty years earlier. The niche formed by an ulcer on the lesser curvature of the duodenal bulb can be recognised in the silhouette of the bulb—one on the anterior or posterior wall can be recognised only when a radiograph is taken whilst the bulb is being compressed. A test-meal generally shows hyperchlorhydria with a climbing curve and hypersecretion. If a lower curve is obtained, this is generally due to associated gastritis, and is replaced by a high curve if the meal is repeated when treatment has resulted in healing of the ulcer and disappearance of the gastritis.

### Diagnosis

When the symptoms of duodenal ulcer have been present for a short time only an actual chronic ulcer is not as a rule present, but a pre-ulcerative duodenitis, which may be associated with a rapidly emptying duodenal bulb but no constant x ray deformity and with occult blood in the stools. It is likely to result in an ulcer if not adequately treated. Excessive smoking over fatigue and anxiety may give rise to almost identical symptoms in individuals with the hypersthenic gastric diathesis, but the x rays show no abnormality and occult blood is absent from the stools.

The diagnosis from gastric ulcer depends upon the later onset of pain the greater relief on taking food the comparative rarity of vomiting the greater frequency of a climbing hyperchlorhydric curve obtained with a fractional test-meal, the frequent situation of pain and tenderness to the right of the middle line the more frequent periods of complete freedom from symptoms, and the results of x ray examination, which occasionally however reveals the presence of an ulcer in the stomach as well as in the duodenum. Hæmatemesis without melæna is much more common in gastric than in duodenal ulcer and melæna alone is more common in duodenal ulcer. The symptoms may closely resemble those of cholecystitis, but in the latter condition the pain is much less regular in its time relations the tenderness is over the gall bladder and evidence of gall bladder disease is obtained by cholecystography and duodenal intubation.

### Treatment

"It is a hard thing to observe a strict diet yet he that loves himself, will easily endure this little misery to avoid a greater inconvenience (Richard Burton, *The Anatomy of Melancholy* 1621)

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If the ulcer is in the neighbourhood of the pylorus and is giving rise to obstruction continuous hypersecretion of gastric juice will occur throughout the night. It is then impossible for the ulcer to heal, and in the past it has been supposed that such cases require operation. But the obstruction is generally due entirely or in great part to surrounding oedema and inflammatory swelling and pyloric achalasia or spasm and in such cases, if the ulcer can be caused to heal, any scarring produced is insufficient to give rise to obstruction. No feed should be taken after 6 p.m. and at 10 p.m. the stomach is completely emptied by Senoran's evacuator if not more than 4 oz. of fluid are present on two consecutive nights this can be discontinued. In most cases the continued nocturnal secretion is rapidly controlled by this treatment.

The strict treatment should be continued without modification until for at least two weeks the patient has had no spontaneous pain and no trace of tenderness or rigidity no occult blood has been found in the stools, and the x rays no longer show the presence of an ulcer crater. A gastric ulcer should not be regarded as cured until gastroscopy shows that healing is complete, the crater may be filled with granulation tissue so that no niche is seen with the x rays a fortnight or more before it is replaced by mucous membrane. In no case should the strict treatment last for less than four weeks and for large and chronic ulcers eight or twelve weeks may be required. The pain generally disappears within forty eight hours the other signs of activity disappear considerably later, the exact time depending upon the size and age of the ulcer. When healing is complete the diet can be rapidly increased until at the end of three or four weeks the patient passes to the following post-ulcer regime, to which he must adhere for the rest of his life.

#### *Post Ulcer Regime*

(To be followed permanently)

A meal or feed (milk, plain biscuit or plain chocolate) should be taken at intervals of not more than two hours from waking to retiring, and again if awake during the night.

Avoid alcohol except, if desired later on a small quantity of beer light wine or diluted whisky at meals.

Avoid nuts, pips and skins of fruit (raw cooked or in jam) and raisins, currants figs, ginger and lemon peel in puddings and cake.

The patient should be kept warm in bed throughout the treatment, but he should get up every day to have a bath and to open his bowels, the difficulties with which are greatly reduced if a bed-pan can be avoided. The object of dietetic treatment is to reduce the quantity and acidity of the gastric juice and to provide sufficient suitable food to maintain the patient's nutrition, whilst being as un-irritating in its mechanical and chemical characteristics as possible.

The details of the strict diet, modified to fit in with war conditions, are summarised below.

### *Strict Ulcer Treatment*

The patient should remain in bed on the strict treatment without alteration until healing is complete.

Every even hour whilst awake 5 oz. of citrated milk. This can be warm or cold and may be flavoured with tea.

Every odd hour a 5 oz. feed which may be made of any of the following:

(a) Arrowroot, farola, Benger, Horlick, junket, custard. These can be made more appetising by the addition of a little red currant, apple or other fruit jelly, and the junket may be flavoured with chocolate.

(b) At least two should consist of a thick soup or semi-solid purée of potato, artichoke, cauliflower or parsnip.

During the night the patient should have citrated milk by his bedside, so that whenever he wakes he can take a feed.

A rusk, plain biscuit or thin bread with butter or honey may be eaten with any of the feeds. A "coddled egg" may be taken once or twice a day.

Small quantities of water may be drunk between feeds. An ounce of strained orange, tomato, or other fruit juice should be taken with three of the drinks. When unobtainable, 100 mgm. of ascorbic acid dissolved in milk should be given daily.

Ten grains of sodium citrate in a teaspoonful of water should be added to each milk feed.

A drachm of atropine mixture (atropine sulphate, gr.  $\frac{1}{100}$  in 1 dr. water) is given before the last feed and before two or three other feeds if the acidity is high. The dose should be increased by 10 minims every day until an unpleasant degree of dryness of the mouth or paralysis of accommodation occurs, the dose should then be reduced to that of the previous day.

Half a teaspoonful of magnesium trisilicate (magsorbent) may be given with a little water half-way between feeds and a teaspoonful last thing at night.

Wash the mouth out after each feed.

No smoking during the strict treatment.

nervous system. This *faiblesse irritable* gives rise to symptoms in any organ the physiological and anatomical functions of which are less efficient than the average. Consequently it is only individuals with the hypersthenic and hyposthenic gastric diatheses who are likely to develop stomach symptoms as a result of exhaustion. Their symptoms are then very similar in character to those produced by the organic diseases which may be associated with these diatheses.

Depressing emotions and the psychological factors concerned in the production of the anxiety neuroses may also give rise to similar gastric symptoms in the predisposed. They may also be important in the development of gastric and especially of duodenal ulcer anxiety being a common cause of recurrences and of the sudden increased activity of ulceration which may result in hæmorrhage or perforation.

### Symptoms

The gastric symptoms in nervous dyspepsia are characterised by their irregularity the patient feeling very ill one day and comparatively well the next without any obvious reason for the change. The most constant complaint is of vague epigastric discomfort which rarely amounts to actual pain. It is generally worst in the morning and improves later in the day but sometimes the symptoms increase when the patient becomes more fatigued towards evening. The discomfort is aggravated by meals a sensation of fullness being felt as soon as a small quantity of food has been eaten but it is often present to a minor degree all day. It has little relation to the amount or the kind of food, differing in this way from the discomfort of organic gastric disorders. It is increased by worry and excitement, while some new interest, whether it be a change of surroundings, a game, a conversation, a new medicine or a new doctor leads to its temporary disappearance. In contrast with most ulcer cases, in which pain is relieved by both alkalis and food, food differs from alkalis in generally failing to give relief (Edwards and Copeman, 1943). Nausea and vomiting, either separately or together may occur. Many patients complain of flatulence, which is generally due to *aërophagy*. Frequent noisy belching is common, especially among older men. The appetite is often diminished. Constipation is generally present and the patient often aggravates his symptoms by overdosing himself with purgatives.

Radiological reports of hypertonus, hypotonus or gastroparesis

Raw and cooked green vegetables are allowed if very thoroughly chewed, but the coarser varieties should be passed through a cullender. The skin of tomatoes and pickles of all kinds should be avoided. Porridge is allowed only if made with the finest oatmeal.

Avoid vinegar, unripe fruit, pepper, mustard, curry, chutney, new bread, cooked cheese, fried fish, tough meat.

Eat slowly and chew very thoroughly. An adequate time should be allowed for meals, rest whenever possible for at least a quarter of an hour before and after meals. Meals must be punctual. When there is no time for a proper meal, it is better to drink some milk or eat some plain chocolate or biscuits rather than bolt some less digestible solid food.

Do not smoke more than six cigarettes or two pipes a day, and these should be after meals, cigarettes and pipes should have an absorbent plug in their mouthpiece. No smoking at all if any indigestion. It is best to give it up entirely.

A teaspoonful of magnesium trisilicate or aludrox should be taken an hour after meals and also whenever the slightest indigestion or heartburn is felt. If the curve of acidity is very high, the atropine mixture should be taken before meals and last thing at night.

The bowels should be kept regular by means of isogel or liquid paraffin, and no other aperients should be taken.

Have your teeth attended to regularly every six months. If you have insufficient teeth for efficient mastication, wear dentures.

Take no drugs in tablet or pill form. Avoid aspirin and veganin.

Special care should be taken to avoid chills. If you get a cold, sore throat, influenza or other infection, remain in bed on a very light diet until you have completely recovered.

During periods of overwork, and especially of mental stress, whenever possible a day or half a day a week should be spent at rest in bed or lying in the open air, on a strict diet, even in the complete absence of digestive symptoms.

If you have the slightest return of symptoms, go to bed on a strict diet at once. Consult your doctor and do not wait for the symptoms to get serious.

## FUNCTIONAL GASTRIC DISORDERS

### Ætiology

Exhaustion from physical or mental overwork, insufficient sleep, long residence in the tropics, insufficient food, and the toxæmia of infections has a depressing effect on all bodily functions and at the same time it tends to increase the irritability of the visceral

nervous system. This *faiblesse irritable* gives rise to symptoms in any organ the physiological and anatomical functions of which are less efficient than the average. Consequently it is only individuals with the hypersthenic and hyposthenic gastric diatheses who are likely to develop stomach symptoms as a result of exhaustion. Their symptoms are then very similar in character to those produced by the organic diseases which may be associated with these diatheses.

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organic disease such additional investigations as a test-meal, examination of the stools for occult blood and occasionally gastroscopy. He can then be confidently told that he has no organic disease and that with perseverance he will get well. The general nervous condition first requires attention for this a short period of mental and physical rest followed by graduated exercise and sufficient food to overcome the inanition are the chief indications. Congenial surroundings, cheerful companions and appetising food are of great importance. For any associated anxiety symptoms a simple form of psychotherapy is essential. This can be given by any intelligent and sympathetic medical officer more elaborate methods which require the help of a specialist in psychotherapy being rarely required. In a recently published report on the disposal of dyspepsias in soldiers it was stated that if no ulcer is found on radiological examination, the majority are sent for psychological investigation by the military specialists in psychotherapy. Nothing could be more unwise. If a similar arrangement held in civilian life, the general practitioner would send at least 50 per cent. of his patients to the psychiatrist, although he himself is far more likely to make a correct diagnosis and to cure the patient than a "specialist in psychiatry" whose help is required in perhaps 1 per cent. of cases.

The dyspepsia itself generally requires no special treatment, as it disappears with reassurance that there is no organic abdominal disorder and with successful treatment of the underlying nervous condition.

### HYSTERICAL GASTRIC SYMPTOMS

When symptoms such as vomiting, anorexia, nausea and abdominal pain have been caused by emotional disturbance or by gastritis, gastric ulcer, appendicitis or other organic cause, they may continue or recur as the result of suggestion after the original cause has disappeared. They are then hysterical symptoms. Hysterical digestive symptoms are always curable by simple psychotherapy in the form of explanation, persuasion and re-education.

#### Hysterical Vomiting

Acute gastritis was a common symptom in all forms of gassing in the last war. The irritant gas called forth an abundant secretion of saliva and nasal mucus, in which some of it was dissolved and

should be ignored, as these represent variations in the normal length of the stomach and not pathological variations in tone or position. Test-meals show the usual variations found in normal people, 10 per cent. of whom have hyperchlorhydria and 10 per cent. hypochlorhydria. In a paper published in the *British Medical Journal* in July 1941 on dyspepsia in a military hospital 43 per cent. of 124 cases were described as "functional dyspepsia" and ascribed to hyperchlorhydria, hypochlorhydria, hypermotility, gastroparesis or atony, all of which are normal variations from the average, which never give rise to symptoms. When nervous dyspepsia develops in a patient with constitutional hyperchlorhydria, the symptoms are likely to simulate those of duodenal ulcer. The abdominal muscles may be tense so that examination is difficult, but there is never any great degree of tenderness, and what is present is diffuse and variable in position rather than localised and constant.

The gastric symptoms are always associated with other nervous symptoms, such as headache and insomnia, and anorexia may lead to progressive loss of weight and strength. The abdominal discomfort may be accompanied by flushing of the face, palpitation and coldness of the extremities. The patient is generally depressed and pessimistic, he pays great attention to all his bodily functions, frequently looking at his tongue and minutely inspecting his stools. His account of his symptoms is full of details, and he often has some theory to account for them, believing himself to be suffering from some mythical disorder such as acidity, a dropped stomach or colon, mucous colitis or intestinal auto-intoxication, and not infrequently he fears he has cancer.

### Treatment.

In mild cases with a short history, such as are common among recruits, a diagnosis can often be made with a considerable degree of accuracy after a thorough clinical examination without even the help of radiography. Such cases should be treated by their regimental medical officers and not sent into hospital, as with reassurance and an alkaline mixture many men return to duty free from symptoms in four days and the large majority of the remainder after another four days (Leigh, 1941). In severe cases and in those of longer duration the patient's confidence must be gained by sending him to hospital for a more complete examination, including not only radiography, but when necessary for the exclusion of

organic disease such additional investigations as a test-meal examination of the stools for occult blood and occasionally gastroscopy. He can then be confidently told that he has no organic disease, and that with perseverance he will get well. The general nervous condition first requires attention for this a short period of mental and physical rest, followed by graduated exercise and sufficient food to overcome the inanition are the chief indications. Congenial surroundings, cheerful companions and appetizing food are of great importance. For any associated anxiety symptoms a simple form of psychotherapy is essential. This can be given by any intelligent and sympathetic medical officer more elaborate methods which require the help of a specialist in psychotherapy being rarely required. In a recently published report on the disposal of dyspepsias in soldiers it was stated that if no ulcer is found on radiological examination the majority are sent for psychological investigation by the military specialists in psychotherapy. Nothing could be more unwise. If a similar arrangement held in civilian life the general practitioner would send at least 50 per cent. of his patients to the psychiatrist, although he himself is far more likely to make a correct diagnosis and to cure the patient than a 'specialist in psychiatry' whose help is required in perhaps 1 per cent. of cases.

The dyspepsia itself generally requires no special treatment, as it disappears with reassurance that there is no organic abdominal disorder and with successful treatment of the underlying nervous condition.

### HYSTERICAL GASTRIC SYMPTOMS

When symptoms such as vomiting, anorexia, nausea and abdominal pain have been caused by emotional disturbance or by gastritis, gastric ulcer, appendicitis or other organic cause, they may continue or recur as the result of suggestion after the original cause has disappeared. They are then hysterical symptoms. Hysterical digestive symptoms are always curable by simple psychotherapy in the form of explanation, persuasion and re-education.

#### Hysterical Vomiting

Acute gastritis was a common symptom in all forms of gassing in the last war. The irritant gas called forth an abundant secretion of saliva and nasal mucus, in which some of it was dissolved and

swallowed The irritation of the stomach produced a varying degree of inflammatory reaction, which gave rise to vomiting by a protective reflex, the object of which was to remove the irritant from the stomach Post-mortem examination in fatal cases showed the presence of acute gastritis, the existence of which was also demonstrated by the presence of abundant mucus with traces of blood in the material vomited during the first twenty-four hours In the large majority of cases vomiting ceased after two or three days or at the latest by the end of a week When vomiting persisted for more than a fortnight after gassing, it was no longer caused by irritation by the gas but was due to the hysterical perpetuation of the symptom Instead of calling for restriction of diet, lavage and drugs, it could now be rapidly cured by psychotherapy

All of the numerous cases of hysterical vomiting following gassing which came under our care had already been in other hospitals for many months, and the vomiting had been diagnosed as due to gastritis, although it was the only symptom present and the vomited material contained no trace of blood and no excess of mucus. A few had been diagnosed as gastric or duodenal ulcer, and one man had even been subjected to a gastro-enterostomy, which naturally had led to aggravation of his vomiting Several men were pensioners who had been invalided from the service on account of "gastritis," but no improvement had followed their return to civil life Without exception they all recovered rapidly with psychotherapy and were able at once to take a full diet and lead a life of normal activity, in spite of having been on a strict and often purely fluid diet for many months In some cases they had been kept in bed during the entire period

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A few of our cases of hysterical vomiting in soldiers followed vomiting which had developed as a symptom of an acute infection, such as trench fever and dysentery A few also were the sequel of emotional vomiting Thus a soldier was detailed to bury some decomposed German corpses The work was of such a revolting character that he was violently sick, and the vomiting continued for several months until the origin and nature of his symptoms were explained to him and he was taught to control the habit

In February 1941 I saw a highly strung young French-Canadian soldier who had for several weeks been vomiting after every meal or feed even when on a pure milk diet. The vomiting which was not associated with any pain or discomfort was the immediate sequel of the acute gastric irritation caused by accidentally swallowing some petrol from a bottle which he thought contained water. Treatment in the manner described below resulted in the immediate cessation of vomiting, and he was discharged to duty on a full diet at the end of a fortnight.

In our earliest cases we occasionally used the stomach tube as a means of suggestion, but we soon dispensed with this and confined ourselves to treatment by explanation and persuasion. It is best to be alone in the room with the patient after having examined him and come to the conclusion that he has no organic disease. The cause of his vomiting is explained in language suited to his intelligence. He is made to understand that although at the onset it was the natural result of irritation by gas or the result of an emotion, the primary cause is no longer present, and the persistence of the vomiting is due to the fact that he has become convinced that he is suffering from some disease of the stomach which causes vomiting, and consequently he continues to vomit although this is no longer necessary. It is explained to him that his stomach is perfectly healthy and if it is given sufficient to digest in the form of ordinary food instead of nothing but slops, it will do it perfectly well so long as the patient himself is confident that the explanation is correct. He is not left until he is convinced of the truth of what he has been told and has been successful in retaining an ordinary full meal. After this he is given a full diet, and a little encouragement on two or three subsequent occasions is all that is necessary to consolidate the cure.

The most important part of the treatment is its rapidity and the avoidance of any accessories such as isolation, diet or drugs. But it is essential for success that the physician should be confident of his diagnosis and that the patient should have confidence in his physician.

#### Pseudo Flatulence: Hysterical Spasm of Diaphragm

Colonel T. G. Moorhead observed a number of patients in Egypt, in whom severe abdominal distension developed from four to eight months after apparent recovery from dysentery which had in most cases developed in Gallipoli. They complained of a feeling

swallowed. The irritation of the stomach produced a varying degree of inflammatory reaction, which gave rise to vomiting by a protective reflex, the object of which was to remove the irritant from the stomach. Post-mortem examination in fatal cases showed the presence of acute gastritis, the existence of which was also demonstrated by the presence of abundant mucus with traces of blood in the material vomited during the first twenty-four hours. In the large majority of cases vomiting ceased after two or three days or at the latest by the end of a week. When vomiting persisted for more than a fortnight after gassing, it was no longer caused by irritation by the gas but was due to the hysterical perpetuation of the symptom. Instead of calling for restriction of diet, lavage and drugs, it could now be rapidly cured by psychotherapy.

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A few of our cases of hysterical vomiting in soldiers followed vomiting which had developed as a symptom of an acute infection, such as trench fever and dysentery. A few also were the sequel of emotional vomiting. Thus a soldier was detailed to bury some decomposed German corpses. The work was of such a revolting character that he was violently sick, and the vomiting continued for several months until the origin and nature of his symptoms were explained to him and he was taught to control the habit.

billets is insufficient and is frequently filthy, and in spite of the fact that latrines in British camps are generally well looked after and clean, they are often so exposed to the weather that the soldier opens his bowels as rarely as possible when it is cold or rainy. The latrines in a large hospital in Lemnos in November and December 1915 were entirely unprotected from the weather with the result that the seats were generally soaked with rain and occasionally covered with an inch or more of snow. No wonder that the officers, orderlies and patients who were often compelled to open their bowels with abnormal frequency as a result of dysentery tried to avoid opening them at all.

The desire to defecate, voluntarily suppressed in this way for many days, gradually becomes less frequently felt, and finally it may almost completely disappear. The dyschezia thus produced could in the early stages be overcome by a simple effort of will but a man often fails to realise how easily he could open his bowels in spite of feeling no desire to do so, and presumes that it is useless to make the attempt. He therefore takes a strong aperient pill whenever the conditions are not too uncomfortable, especially if he is beginning to feel some of the local or general ill-effects of the retention of faeces in the rectum. This leads to such violent peristalsis that the fluid faeces in the proximal part of the colon are rushed into the rectum from which they are discharged, together with the hard faeces which have accumulated in it.

By the time they return to normal conditions of life many men have become so accustomed to their periodic pills that they continue to use them and never make an effort to defecate without their aid. This habit is often confirmed when a man is taken into hospital for a wound or disease as he can almost invariably obtain a pill for the asking and is very rarely taught how to get well without artificial aid. The Seale Hayne Hospital was, I believe, almost the only military hospital in the country during the last war where the nurses were not allowed to give pills unless they had been ordered by the medical officers, where the medical officers ordered aperients only in most exceptional cases, and where pills and salts could not be bought at the canteen.

The final condition is thus often one of hysterical dyschezia, as it is caused by auto-suggestion, the patient having convinced himself that he cannot open his bowels without aperients. It is often aggravated by the hetero-suggestion produced by being given

of abdominal distension with dyspepsia and dyspnoea. The bowels were regular. Nothing abnormal was found on examination except enormous abdominal distension, which was sometimes so great that ascites was simulated, and in two cases an attempt had been made to tap the abdomen before Moorhead saw the patients. I saw a similar condition in a man invalided from Salonica with gastritis and diarrhoea, his abdomen was the size of a seven-months' pregnancy. X-ray examination showed that the abdominal protrusion was not the result of distension, but of spasm of the diaphragm. Since that time I have seen many cases in civil life and two in soldiers in the present war. Several of the civilians had undergone exploratory laparotomy, sometimes more than once, for what was supposed to be intestinal obstruction, in spite of the disappearance of the distension under the anaesthetic.

In some cases the condition appears to be a primary psychoneurosis. In others it is a complication of some organic disorder, such as a gastric ulcer or colitis, and it may follow a blow on the abdomen. The spasm may be continuous and last for weeks or months. More frequently it occurs in attacks, the distension then disappears as suddenly as it comes without eructation or passage of flatus. The condition is easily recognised by the sharp drawing in of the lower ribs which results from the pull of the contracted diaphragm. This is in striking contrast with the expansion of the lower part of the thorax, which is present when the abdomen is distended with fluid or dilated intestines. X-ray examination shows that there is no excess of gas in the stomach or intestines, and that the diaphragm is very low and its movements are shallow. The condition can generally be quickly cured by vigorous manipulation of the abdomen and teaching the patient control of his diaphragmatic movements, but sometimes it is very resistant to treatment.

#### CONSTIPATION IN SOLDIERS

The conditions of active service may lead to the voluntary suppression of defæcation, with the result that dyschezia is extremely common. In the fighting line the irregular hours for sleep and meals, the difficulty of getting to a latrine without exposing one's self to danger, together with the insufficient shelter from the weather and frequent insanitary surroundings of the latrine itself make regular defæcation almost impossible. Behind the lines conditions are often very little better. The lavatory accommodation in most

## CHAPTER XVII

### EFFORT SYNDROME

#### SOLDIER'S HEART      DISORDERED ACTION OF THE HEART

The effort syndrome is a condition in which the symptoms and signs produced in healthy people by excessive exercise are called forth by an effort which should normally give rise to no unpleasant symptoms and in which no physical signs of organic disease are present. The smaller the effort required to produce these symptoms and signs the more severe is the disorder. The effort syndrome occurs most frequently among soldiers it is rare in the Air Force and very rare in the Navy. It has been a much less important problem in the present war than in 1914-18. It develops during the period of training but more often during active service. The symptoms differ in no way from those which may occur among civilians but their relative frequency led to the use of the term 'soldier's heart' to describe the various functional cardiac disorders specially common among soldiers. In the last war the condition was known as D.A.H. or disordered action of the heart to distinguish it from V.D.H. or valvular disease of the heart. As it is most undesirable that patients should have their attention drawn to their heart, the term "effort syndrome," suggested by Lewis in 1917 has now been officially adopted. The effect of active service on the heart was first investigated during the American Civil War by Harthorne (1864) and da Costa (1871) and the last and the present war have given opportunities for much research on the subject.

The importance of the effort syndrome in the last war can be gathered from the fact that 2,503 out of 33,919 or 7.4 per cent. of the soldiers and sailors invalided from the services from the beginning of the war up to May 31st 1916 were discharged on account of heart disease and by May 1918 the number had risen to 38,569. The problem of invaliding on account of soldier's heart became such a serious one that the War Office and Medical Research Council arranged for the segregation of heart cases at the Hampstead Hospital under Thomas Lewis. Later the work was transferred to Colchester and finally centres were established

aperients, and it can almost invariably be removed by the psychotherapy involved in explaining to the patient the nature of his supposed constipation, prohibiting the use of aperients, and re-establishing the conditioned reflex upon which normal defæcation depends by insisting that an attempt should be made at the proper time and under proper conditions, even in the absence of any "call to defæcate" In some cases it is necessary to empty the rectum by a plain water enema as a preliminary to effective psychotherapy, as the accumulation of fæces may be so large that it is difficult for the dilated rectum to empty itself by means of a simple effort

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(ii) *Feeble development resulting from sedentary occupation*.—Out of 543 patients suffering from effort syndrome Lewis found that 306 or 57 per cent. were recruited from sedentary or light occupations. Some of them had given up heavier work on account of similar but less severe symptoms which disappeared when they lived a less strenuous life and took comparatively little exercise. But apart from these individuals, who were apparently affected with the condition before they joined the Army men who have followed a sedentary occupation are much more likely to break down during training than those who have previously done heavy work, unless special care is taken in the early days of training, as their hearts are less developed and have less reserve power.

(iii) *Congenital or acquired organic disease*.—Of the few men with definite evidence of congenital or acquired organic heart disease, who escape the vigilance of the recruiting medical officer the majority break down during training and are invalided from the service. But if the myocardium is unaffected, valvular disease may be so far compensated that the strenuous life of a soldier on active service is possible. Several cases were observed, in which soldiers carried out their duties satisfactorily at the front for many months, and a valvular lesion was discovered accidentally only on admission to hospital for a wound or other cause. There is, however, no doubt that a man with valvular disease of the heart has less reserve power than a man with a normal heart, and any infection which he contracts is particularly likely to affect his heart, so that he develops symptoms when he tries to resume his former duties, and there is the further risk that he may develop subacute bacterial endocarditis. Whenever old valvular disease is accidentally discovered in a sick or wounded man he should be reclassified for home service, though there is no reason why he should be invalided from the army if he has had no symptoms of cardiac insufficiency. A history of rheumatism or chorea in childhood is often obtained in cases of effort syndrome in which there is no evidence of valvular disease. It is clear that the myocardium must have been weakened, and this naturally predisposes to the development of cardiac symptoms under the strain of active service.

(iv) *Old heart strain*.—In many cases the patient admits that he has had similar but generally less severe symptoms in the past after taking unwonted exercise. Officers not infrequently say that they suffered from somewhat similar symptoms when

for heart cases in each home command. At the same time it became recognised in France that many cases were being sent to England with an erroneous diagnosis of valvular disease of the heart and still more with trivial cardiac symptoms. In 1916 all cases sent to one base diagnosed as V D H and D A H. were collected in a single centre under Colonel W. E. Hume, and in 1917 similar centres were started at four other bases. At these centres treatment of D A H was so successful that 50 per cent of cases were detained for duty in France, but far too many escaped diagnosis and reached England without having passed through any heart centre. In the present war it has been found necessary to establish no more than two such centres in England and one in Scotland. The total admissions into the largest of these was only about 700 up to May 1941 (Wood).

### Ætiology.

Effort syndrome is most commonly due to the effect of over-exertion, often associated with prolonged mental strain and insufficient sleep, on a heart and nervous system, which were already weak before the war, or which have become weakened on active service as a result of some form of toxæmia. Although physical strain, anxiety and toxæmia generally act together, in many cases the first or the last can be definitely excluded, but a psychological factor is always present and is sometimes alone responsible for the development of the syndrome.

(a) **Feeble Circulation, Congenital or Acquired before the War.** (1) *Congenital cardiac insufficiency*—A man may be born with a circulation which is sufficient for ordinary purposes, but which has a deficient amount of reserve power for increased strain. In such cases the peripheral circulation has generally been feeble from early childhood, as shown by the cold, damp, blue extremities, small pulse and distended veins. The chest is often narrow and badly developed, and the heart is small. The circulation is unable to cope with strenuous efforts, and the pulse becomes rapid on slight exertion. Individuals of this kind, having learnt their limitations, generally adopt a sedentary occupation in civil life and do not indulge in out-of-door sports. They are very liable to break down during military training, but if sufficient care is taken, they may become efficient soldiers, though they remain abnormally liable to develop the effort syndrome under the conditions of active service.

80 per cent greater than in the general population over the same period, and that those who originally showed abnormal but indicative signs in the lungs were three times more liable to develop the disease than the remainder of the group. A history of venereal disease is rare. The improvement which followed the removal of septic teeth was sometimes so marked that it is probable that they were responsible for poisoning the heart muscle. When the effort syndrome is once present any intercurrent infection is very likely to aggravate the symptoms.

(ii) *Excessive smoking*—I am convinced that the toxæmia which results from excessive smoking is often a very important factor but it is not an essential one, as many cases were observed in non-smokers and the condition was as common among Sikhs who do not smoke as among other Indian troops (Macgregor). Many soldiers, especially those under twenty years old, smoked much more than they did before they joined the Army and the custom of giving cigarettes to convalescent soldiers whilst they are in hospital recovering from various infections is responsible for much subsequent trouble, as the heart and nervous system, already poisoned by the toxins produced by the infection, are particularly liable to be further damaged by the toxins inhaled whilst smoking. Some patients discover for themselves that their symptoms are aggravated by smoking and spontaneously reduce their allowance of cigarettes.

Parkinson and Koefod (1917) investigated the immediate effect of smoking five cigarettes in forty minutes on the circulation of forty smokers, of whom thirty were suffering from effort syndrome and ten were healthy soldiers. The pulse-rate was raised an average of nine beats during the smoking in the former class and six in the latter and the blood pressure was raised by 5 to 10 mm. Hg in both. No irregularity was produced, and respiration was unaffected. Two patients complained of precordial pain. The pulse-rate was more affected by exercise after smoking than before in the patients, but not in the controls, and half of the former but only two of the latter were more breathless. Parkinson and Koefod concluded that excessive smoking often increases the breathlessness and precordial pain in the effort syndrome.

(iii) *Alcohol*.—The statistics collected by Lewis revealed the unexpected fact that 53 per cent of 454 patients suffering from effort syndrome were abstainers, and that those who are abstainers

fifteen or sixteen years old after too strenuous indulgence in long-distance or cross-country running. This has often been regarded as "heart strain," but careful inquiry generally shows that the heart was probably already in a weakened condition owing to some slight infection which immediately preceded the supposed strain or which was actually present at the time. Allbutt long ago pointed out that so-called heart strain is almost always due to the effect of stress upon hearts already enfeebled by infections.

(b) INTOXICATION (1) *Toxæmia from infections*—The toxæmia is most frequently bacterial in origin, the symptoms dating from some preceding infective disease. This is sometimes a definitely recognised infection. Some cases have followed rheumatic fever, apart from the 12 per cent (compared with 5 per cent in slightly wounded controls) in which a history of rheumatism or chorea in childhood is obtained, but these should probably be regarded as examples of organic myocardial disease, although the symptoms may be indistinguishable from those of the effort syndrome. Dilatation of the heart and tachycardia may develop in the course of typhoid and paratyphoid fever. Epidemic catarrhal jaundice was a common cause of effort syndrome at Gallipoli, though this complication is not observed after infective and spirochætal hepatitis. Bacillary and amœbic dysentery may be followed by cardiac weakness, but less frequently than the non-specific intestinal disorders associated with diarrhoea and little or no rise of temperature, in which the nature of the infection is obscure. A history of recent malaria, diphtheria, recurrent tonsillitis, pneumonia or bronchitis, and occasionally of measles or scarlet fever may be obtained. Trench fever was probably the most common infection to be followed by effort syndrome in the last war. It frequently escaped recognition, being labelled as P U O or as influenza, which was rare in the Army until the pandemic at the end of 1918. In some cases the infection is not sufficiently severe to cause the soldier to report sick, and keen men often continue at duty in spite of having a febrile illness, which ought to be treated by complete rest. Residual sepsis from wounds was a further cause of myocardial poisoning in many cases. According to Weil (1916), latent tuberculosis, whether of recent origin or of long standing, was a common cause of cardiac symptoms in French soldiers. The investigations of Grant (1925) on the after-history of men suffering from the effort syndrome showed that the incidence of pulmonary tuberculosis was

effects, are sufficient to cause nervous exhaustion and cardiac weakness.

(d) THE NERVOUS FACTOR. From investigations carried out at the Military Heart Hospital, Colchester Oppenheimer and Rothschild (1918) found a family history of neuroses in 56 per cent. of cases compared with the 78 per cent. found by Wolfsohn among soldiers suffering from war neuroses and 38 per cent. among wounded. Although there was no family history of insanity or epilepsy in any of the wounded, this was present in 23 and 15 per cent. respectively among the cases of effort syndrome. The percentage with a personal history of neuroses was 51 contrasted with 76 among cases of war neuroses and 12 among the wounded, and of those with a positive family and personal history there were 46 per cent. among the effort syndrome and only 6 per cent. among the controls. Of those with a bad family or personal history only 4 out of 48 did any full military service in contrast with 33 out of 39 with a good history. Among the former the breakdown generally occurred without any obvious reason, but among the latter it followed some infection, gassing, exposure to a shell explosion, or prolonged service in 20 out of 46 cases. The former group generally suffered from precordial pain, whereas the latter had marked fatigue symptoms but rarely much pain.

Effort syndrome is often nothing more than a manifestation of an anxiety neurosis. But it is especially likely to occur when anxiety is associated with toxæmia and physical fatigue. The latter give rise to slight circulatory disturbances, which would often be insufficient to produce any subjective symptoms in the absence of the anxiety state. The nervous condition of the patient leads to the unconscious exaggeration of his symptoms, which in time becomes a further cause of anxiety as they lead to the fear of serious heart disease. It is exceptional for the effort syndrome to be a pure neurosis, though it appeared to be so in the following three cases.

A previously healthy soldier was blown up by a shell in November 1914. He did not lose consciousness but his heart at once began to palpitate and his breath became rapid and laboured. He had never before suffered from similar symptoms. His pulse continued to be rapid and the cardiac impulse was very forcible. The least excitement or exertion caused palpitation, tremor of the eyelids and to a less extent of the hands and flushing of the face. No improvement occurred even after he was invalided from the Army in May 1916. I first saw him in August 1916. Under hypnosis his heart became

or drink very little do considerably worse than those who drink more heavily. He suggested that this is due in part to the greater frequency of abstinence among men following sedentary occupations than among the stronger men who live a more active life.

(iv) *Cordite poisoning*—In the South African War many soldiers purposely produced cardiac symptoms by eating cordite. With the exception of a few cases observed in 1914, this was a very rare occurrence in the last war, and it has not been recorded in the present war.

(v) *Gas poisoning*—Cardiac symptoms frequently followed exposure to chlorine and phosgene gas and less frequently mustard gas in the war of 1914–18 (p. 497).

(vi) *Endocrine type*—In a small proportion of cases there is evidence of functional over-activity of the thyroid and suprarenal glands following prolonged nerve strain (*vide* p. 143). Hyperthyroidism due to organic hyperplasia of the thyroid may give rise to circulatory symptoms, but it is very rare in soldiers, and the clinical picture is quite different from that of effort syndrome. Cases have been reported of men taking thyroid pills in order to escape service, but this form of doping does not appear to have been practised after enlisting.

(c) **OVER-EXERTION** Over-exertion is a relative term. A well-trained man can do work, which would be impossible in the early stages of his training and would again be impossible if his heart and nervous system become damaged by the toxins produced by an infection or excessive smoking. Effort syndrome in the recruit, which was at one time ascribed to restriction of the thoracic movement by the accoutrements and at a later date to a badly devised form of drill, but continued to occur with undiminished frequency after the accoutrements and drill had been altered, is to a large extent a result of attempting to train too rapidly. It is as common in the big guardsman who has outgrown his strength as in the under-developed and under-fed recruit from the slums. Skilful and graduated training would prevent its development in peace time as in war.

The trained soldier probably never develops cardiac symptoms as a result of over-exertion whilst on active service unless a toxic factor is also present. But with the nervous system and heart enfeebled by toxæmia, the physical fatigue and mental strain associated with active service, which hitherto produced no ill-

timed insult. But fortunately he restrained himself. The palpitations and the sweating and the trembling however recurred whenever he was with an officer especially that officer. This was noticed and he was asked what was the matter. Not considering it tactful to reply

Because I want to bash you Sir he said he just felt bad and trembly. He was sent to the M.O. an officer in whose company the symptoms persisted. He was put in a shore hospital in Africa. A physician there demonstrated him before five students as a case of Graves's disease saying that he would never be well until he had his thyroid out. This gave the marine food for thought. He was obviously really ill. He began to suffer from choky feeling pain round the heart and thought perhaps he had heart trouble too. Fortunately no surgeon could be persuaded to attack his throat and during the next four months he gravitated slowly through many hospitals, to England.

His mental state when I saw him could be summarised as one of pent up fury. An opportunity to tell his story general appeasement and release of some of his anger helped him greatly. It was at first difficult to persuade him that he was not really ill but demonstration as a case not suffering from Graves's disease or heart disease in front of fifteen students, finally persuaded him of his healthiness. There were many other difficulties about which he needed help but simple psychotherapy on the above lines enabled him to return to duty within three weeks with his aggression directed to more useful channels, and unlucky would be the German foe who should cross his irate path."

Prolonged nerve strain may give rise to circulatory symptoms through the intermediation of the ductless glands, as described in Chapter XIV (p 143)

### Symptoms

The onset of symptoms often dates from some febrile attack, the patient having returned to duty before he felt completely fit. In many cases actual cardiac symptoms are preceded, often for some weeks, by nervous symptoms, which are the result of mental strain associated with toxæmia and fatigue. Physical and mental exhaustion occur very readily and headache lassitude irritability insomnia and tremor of the hands are common. The patient feels unfit even when at rest, and he is often greatly depressed about his health. Excessive sweating, especially of the palms of the hands and soles of the feet is a frequent symptom. Salivation may also occur.

The respiratory rate at rest is about double the normal and the depth is considerably reduced. Ability to hold the breath is much diminished. A moderate amount of exercise which would formerly have produced no ill-effect, gives rise to shortness of breath and

rapidly more quiet, until after five minutes it appeared to be perfectly normal. Repeated suggestion under hypnosis and graduated exercises resulted in rapid recovery.

A soldier complained of severe paroxysmal attacks of tachycardia, especially at night. On cross-examination he admitted that they were invariably caused by a dream or the sudden recollection when awake of the horror he had experienced a year and a half before, when he had found the dead body of a man in his billet.

A man, aged 24, came before a military recruiting board in April 1940. He had never suffered from dyspnoea or distress on exertion and was able to do the heavy work of a gardener without difficulty. He had played football until he was 22, when he gave it up for want of time. On examination he was found to have fairly frequent extra-systoles, but his heart was of normal size and there were no murmurs. Because of the extra-systoles he was certified as being unfit for military service. As a military recruiting board is compelled by the regulations to tell a man why he is rejected, he was given a note stating that he had "disordered action of the heart." The same night his sleep was disturbed by palpitation, and the following day he felt short of breath and experienced discomfort over his heart so that he was unable to work, although he had never before experienced any of these symptoms and had been quite unaware of his cardiac irregularity. His condition did not improve, and from the day of his medical board until his admission into hospital eight months later he did not do a stroke of work, although by that time the extra-systoles had disappeared.

The following admirable description of a purely psychogenic case was written by Felix Browne (1941), who very properly criticises the application of the term effort syndrome to an iatrogenic—doctor-produced—disorder of this kind.

*"The Marine who was Angry"* I was called to see a 24-year-old marine, a burly well-built fellow, complaining of tremor, palpitations, pain over his heart and excessive sweating. Instead of diagnosing effort syndrome, however, I had taken a history which led to a rather different conclusion. Healthy and somewhat hot-tempered, he had carried out his duty on board his ship in the Indian Ocean until 5 months previously. He had lent £4 to a mate who had failed to return it. A fight resulted. He was brought before the captain who said that not only had he disturbed the ship with brawling but had also broken regulations by lending money. Discipline must be preserved, he would therefore forfeit the money and do ten days C.B. The marine did his punishment, but with black rage in his heart. On ending it he went on parade. The officer inspecting stopped opposite him and said, "Look at this silly fellow. What do you mean by coming on parade with long hair like a girl?" The marine trembled, his heart raced, he sweated from every pore in his body, he grasped his rifle, he raised it a little from the ground to bash the officer for such an ill-

timed insult. But fortunately he restrained himself. The palpitations and the sweating and the trembling however recurred whenever he was with an officer especially that officer. This was noticed and he was asked what was the matter. Not considering it tactful to reply "Because I want to bash you Sir" he said he just felt bad and trembly. He was sent to the M.O. an officer in whose company the symptoms persisted. He was put in a shore hospital in Africa. A physician there demonstrated him before five students as a case of Graves's disease, saying that he would never be well until he had his thyroid out. This gave the marine food for thought. He was obviously really ill. He began to suffer from choky feeling pain round the heart and thought perhaps he had heart trouble too. Fortunately no surgeon could be persuaded to attack his throat and during the next four months he gravitated slowly through many hospitals to England.

"His mental state when I saw him could be summarised as one of pent-up fury. An opportunity to tell his story general appeasement and release of some of his anger helped him greatly. It was at first difficult to persuade him that he was not really ill but demonstration as a case not suffering from Graves's disease or heart disease in front of fifteen students, finally persuaded him of his healthiness. There were many other difficulties about which he needed help but simple psychotherapy on the above lines enabled him to return to duty within three weeks with his aggression directed to more useful channels and unlucky would be the German foe who should cross his irate path."

Prolonged nerve strain may give rise to circulatory symptoms through the intermediation of the ductless glands as described in Chapter XIV (p. 143)

### Symptoms

The onset of symptoms often dates from some febrile attack, the patient having returned to duty before he felt completely fit. In many cases actual cardiac symptoms are preceded, often for some weeks, by nervous symptoms, which are the result of mental strain associated with toxæmia and fatigue. Physical and mental exhaustion occur very readily and headache, lassitude irritability in somnia and tremor of the hands are common. The patient feels unfit even when at rest, and he is often greatly depressed about his health. Excessive sweating, especially of the palms of the hands and soles of the feet is a frequent symptom. Salivation may also occur.

The respiratory rate at rest is about double the normal and the depth is considerably reduced. Ability to hold the breath is much diminished. A moderate amount of exercise which would formerly have produced no ill-effect gives rise to shortness of breath and

exhaustion, which may be accompanied by giddiness and faintness. Giddiness may also occur on suddenly standing up. In some cases it is associated with a fall in blood pressure, which is greater than occurs normally under the same conditions. The patient may actually faint after a fright or without obvious cause. The pulse becomes imperceptible, the heart beating at a rate of about 50, when it can be felt again the systolic blood pressure is found to be as low as 60 mm, the rate and pressure rising slowly to normal in the next half-hour. As Lewis points out, the slow pulse and low blood pressure suggest that the attacks are vagal in origin.

More violent exertion causes dyspnoea and palpitation, which may last for some hours. In severer cases the patient gets out of breath after gentle exercise, and palpitation may be troublesome even when he is at rest, especially on first getting into bed at night. The breathlessness manifests itself in a greatly accelerated respiratory rate, exertion which would hardly produce any effect in normal individuals increasing the rate to 40, 50, 60 or even 70 a minute, and ten minutes or more may elapse before the rate returns to normal. All the accessory muscles of respiration come into action, and the reality of the distress is shown by the anxious expression of the patient. The vasomotor centre is frequently affected, so that the hands and feet are always cold. At one time the fingers are shrivelled, white or blue, and numb, at another, they are swollen, red and tingling. The white mark caused by pressure on the skin of the hand takes an abnormally long time to recover its colour.

Occasionally slight swelling of the ankles is present, but oedema is never a prominent symptom. Discomfort, which may amount to severe pain, is generally felt in the left inframammary region, not only on exertion, but also when at rest. It is often a cause of intense worry to the patient, who thinks his heart must be seriously diseased. His anxiety tends to aggravate his pain. The pain was said to be frequently associated with tenderness of the chest wall, the area round the apex might be also involved or it might extend over the greater part of the left side of the chest. Observations on newly admitted cases, however, convinced me that the tenderness was generally, if not always, a result of suggestion by the observer, it was never mentioned by the patient himself, who was unaware of its existence until he was examined.

In some cases, especially when the condition is in part due to

excessive smoking, there is persistent tachycardia. In primarily nervous cases there may be paroxysmal or persistent tachycardia. More frequently the pulse is normal or only slightly increased in rate whilst resting but the least exertion or excitement unduly accelerates it, and several minutes elapse before it returns to normal. Sitting up in bed or standing may change a pulse of 70 to one of 100. Lewis has pointed out that if a patient with effort syndrome and a normal individual do enough work to produce the same degree of dyspnoea, the former of course doing much less than the latter the pulse rate and blood pressure rise on an average to the same height in both. The blood pressure is normal or only slightly raised when at rest except in the endocrine type which is constantly accompanied by hypertension. The pressure however is frequently high if the patient is examined when up, owing to the exaggerated response to exercise and excitement. The influence of emotion was well seen in a patient I examined with Captain G. H. Hunt. His blood pressure for some weeks had constantly been about 160 mm. Hg and pulse rate about 80. On each of two occasions on which he was hypnotised the pressure rapidly fell to 115 mm. and the rate to 68 both gradually rose to their original level after he woke up though he was still lying quietly in bed. He was of a nervous temperament, but was not frightened or excited on the numerous occasions when his blood pressure had been measured. In another apparently similar case there was no fall of blood pressure, though the patient was as deeply hypnotised.

By measurement of the distance of the maximum apex beat from the middle line and by percussion it was never possible to demonstrate any change in the size of the heart (Hume). Orthodiagraphic measurements by Meakins and Gunson (1918) confirmed the views of most clinicians that there was no enlargement of the heart either at rest or immediately after exertion, and Wood (1942) found no enlargement of the heart in any of the 500 cases of effort syndrome he had screened. In at least one large group of base hospitals, however the medical officers were directed to pay great attention to increased dulness to percussion to the right of the sternum, which was believed to be characteristic of the heart in patients convalescent from trench fever. Many men were kept in bed on account of this for long periods and were eventually sent to England as cases of D.A.H. By this time the idea of serious heart disease was so ingrained in their minds that recovery sufficiently complete

for a return to full duty was almost impossible. Many months of hospitalisation were generally followed by invaliding from the service, after which they continued for long periods to complain of anxiety symptoms.

A systolic murmur is frequently heard at the apex and in the pulmonary area, the latter is sometimes very loud and widely propagated. The presence of these murmurs often led to the diagnosis of valvular disease of the heart (V D H.), which had a very unfortunate effect on the mind of the patient. Out of 1,000 cases of cardiac disorders seen by Hume at a convalescent camp in France 169 were sent in as cases of V D H., but in only 55 of them did this diagnosis prove to be correct. The murmurs are always systolic, and are generally much louder in the recumbent than in the erect position, in some cases they disappear completely on standing. They vary more with deep respiration, are diminished to a greater extent by pressure of the stethoscope, and are more exaggerated by emotions and after exertion than murmurs caused by valvular disease. The researches of Lewis and his colleagues at Hampstead showed that the importance of murmurs had been very much exaggerated in the past. Among 462 men suffering from effort syndrome 196, or 42 per cent, had systolic murmurs. Of the latter 49 per cent improved sufficiently to be discharged from hospital to some sort of duty, the proportion being only 42 per cent among those with no murmurs. Lewis concludes that "the presence or absence of systolic murmurs is of no value in estimating the soldier's capacity for work, irrespective of the character, conduction, or point of maximum audibility of the murmur." However true this may be for men who have been admitted to hospital suffering from the effort syndrome, it would be a mistake to extend it to soldiers in general or as a guide in the examination of recruits, as it is doubtful whether 1 per cent. of healthy soldiers have a murmur, whereas one is present in over 40 per cent of men suffering from effort syndrome. There can be little doubt that a man with a "functional murmur," even if he is at the moment suffering from no symptoms of any kind, is much more likely to develop effort syndrome whilst training or under the stress and strain of active service than a man without a murmur.

The electrocardiogram is normal in the effort syndrome and remains so after effort, as was first demonstrated by Parkinson in 1916.

## Diagnosis

The functional disorders grouped together as effort syndrome must be differentiated from the three organic conditions which may cause cardiac symptoms in young men—subacute bacterial endocarditis, chronic valvular disease and angina—the first and especially the last of these are however very rare in soldiers. In functional disorders the temperature is generally normal although there is often a history of an earlier pyrexial attack, and in some cases the infection is so chronic that a slight degree of pyrexia continues for many weeks, or recurrent short attacks of a slight degree of fever occur. In subacute bacterial endocarditis, on the other hand, the temperature is either constantly or intermittently raised. Whereas any murmurs which may be present in the effort syndrome are always systolic, and tend to become less marked as time goes on and the patient's condition improves, diastolic as well as systolic murmurs are often present in infective endocarditis, and new ones may develop owing to organic changes occurring in the valves—they tend to vary in character from week to week and to become more and more marked. Symptoms of infarction in various organs may occur and the general condition of the patient is more serious. In chronic valvular disease there is almost always a history of true rheumatic fever, chorea or occasionally of scarlet fever. Murmurs may be diastolic as well as systolic—they do not become fainter but may actually become louder as the patient's condition improves. More or less hypertrophy is present, which varies with the exact lesion, and the murmurs are as loud in the erect as in the recumbent position—the systolic apical murmur is traceable further into the axilla and often to the back, and pulmonary systolic murmurs are rare except in congenital heart disease. In effort syndrome a feeling of exhaustion often precedes the cardiac symptoms, whereas in chronic valvular disease dyspnoea is generally the first symptom and oedema is much more common. Angina is rare in young men, in whom it is almost always due to syphilis. Pain, which is generally the only symptom, is substernal and not over or below the heart as in effort syndrome in which, moreover the characteristic radiation does not occur. Grant's report on the after history of men suffering from the effort syndrome shows that the presence of even doubtful pulmonary signs should raise the question of tuberculosis, which could nowadays be definitely settled by radiological examination.

**Prognosis.**

Men of feeble physique, who even in civil life have been unable to take much exercise without becoming dyspnoeic and experiencing pain in the cardiac region, and have never been really fit from the time they joined the Army, are unlikely to improve sufficiently to make useful soldiers. With this exception the prognosis is good even in apparently severe cases, and very few men should be invalided out of the Army for the effort syndrome. Complete recovery requires treatment for a period varying between a fortnight in the mildest cases and three to nine months in severe cases.

The disposal of 1,000 men after treatment in a cardiac centre in France in 1917 was as follows (Hume) .

Fit . . . . .	580
Permanent base duty—	
Ordinary . . . . .	172
Light . . . . .	44
Temporary base duty . . . . .	86
Hospital . . . . .	50
England (unfit) . . . . .	6
Unknown . . . . .	17
Still in camp . . . . .	45

Relapses were surprisingly rare only 3 per cent of the patients passed more than once through any heart centre

The prognosis in patients who were sent to England and admitted to the special hospitals at Hampstead and Colchester was far less satisfactory This was due in part to the fact that most of the slighter cases recovered in France or in English hospitals before being transferred to the special cardiac hospital On reaching the latter most patients had had symptoms for a considerable period, and many had become thoroughly convinced that they were suffering from incurable cardiac disease In 1925 Grant published a report of the after-histories of 601 men followed from five to seven years after admission to Hampstead and Colchester Military Heart Hospitals between 1916 and 1918 for effort syndrome Of these 15.3 per cent recovered completely, 17.8 per cent improved, 56.2 per cent remained stationary and 3.2 per cent became worse, 8.7 per cent developed definite disease, most frequently pulmonary tuberculosis, and 2.3 per cent died The number of deaths did not exceed that of the general population

The response to graduated exercise is the best guide to both the immediate and ultimate prognosis. The presence or absence of a systolic murmur is of no value as a guide. Grant (1925) found that only 6 out of 601 cases developed cardiac disease in the following five years and that this small incidence of 1 per cent. was no greater among the 335 with cardiac physical signs than among the 266 others. The occurrence and long continuance of tachycardia and dyspnoea after very slight exertion indicate that the case is severe and the immediate prognosis so bad that the man is unlikely to be of any further use in the Army although ultimate recovery often occurs. The chance of complete recovery steadily diminishes with increasing age. Grant's investigations of 601 men followed for five years after discharge from hospital showed that the proportion restored to good health fell from 25 per cent. in the 17 to 20 age group to 2 per cent. between the ages of 41 and 50 while the percentage of those remaining stationary increased from 51 to 65. The incidence of organic disease and of deaths also increased with advancing years from 3 and 1 per cent. respectively in the youngest to 17 and 6 per cent. in the oldest group.

### Prophylaxis

Feebly developed recruits, who have hitherto followed a sedentary occupation and have taken comparatively little exercise, should not be trained too rapidly. Skilful handling of recruits results in many men, who would otherwise soon complain of cardiac symptoms, becoming strong and capable of considerable endurance. Soldiers suffering from any febrile disorder or from the slightest cardiac disturbance should not be allowed to smoke until they have completely recovered—and then only in strict moderation. Care should be taken not to allow convalescents from infective disorders to get up too soon, but it is equally important that they should not be kept in bed too long. Convalescence should be followed by carefully graduated physical training under medical supervision till the men are fit for full duty. It is of the utmost importance that a diagnosis of V.D.H. should be made only when there is no possibility of mistake about it. The old official diagnosis of D.A.H. or "disordered action of the heart" has very wisely been abandoned, as when the word "heart" has once been mentioned it is often difficult to eradicate the idea of heart disease from the soldier's mind. The fully established condition is now officially recognised as the "effort syndrome" which may however suggest to the

patient that he is incapable of making an effort. For this reason I believe that some such diagnosis as "debility" or, for post-infective cases, "convalescence" is preferable. These are perfectly correct, as there is no doubt that the functional capacity of other parts of the body as well as the heart is impaired.

### Treatment

The first essential in the treatment of the effort syndrome is to encourage the patient and to convince him by explanatory conversations suited to his intelligence that his heart is not really diseased and that he will completely recover his health. The kind of psychotherapy he requires is described in the chapter on the anxiety neuroses (p. 149).

In the acute stage rest in bed may be necessary, but this should never be prolonged. Even if the pulse is still rapid, the patient should be allowed to get up after a short initial period, so long as his temperature is normal. He should be given graduated exercises, which should always be just insufficient to cause exhaustion, dyspnoea or pain. Walking exercises first on the level and then on hills of increasing steepness are of great value. Major C. H. Benham organised graduated exercises of this sort for soldiers with functional cardiac disorders at a general hospital in Salonica in 1916 and the results were very satisfactory, and Colonel W. E. Hume later had a similar experience on a much larger scale at a convalescent camp in France. By the end of the war there was a centre for the treatment of effort syndrome at each of the bases in France. Treatment of a similar kind was also carried out at the special centres in England. In the mornings organised exercises in the open air under an instructor and accompanied by a band should be instituted, they should be followed in the afternoon by games. Officers should be encouraged to golf, shoot or ride, whichever they prefer. Patients should be allowed to rest for a time if they feel tired, out of breath or otherwise unwell. They should be given a regular occupation, such as gardening, farm-work or carpentering for a prescribed length of time every day in addition to their drill and games. A man should not be sent back to his unit until he is able to do an average day's work.

Cardiac stimulants, such as digitalis, do no good and may do harm. Sodium bromide, gr. v, or phenobarbitone, gr.  $\frac{1}{2}$ , should be given two or three times a day in the numerous cases in which

the nervous system is irritable. A small dose of some hypnotic, such as barbitone, will generally procure sleep if insomnia is present

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## CHAPTER XVIII

### TRENCH FEVER

Trench fever is said to have appeared for the first time during the war of 1914 to 1918, and to have disappeared, never to return, soon after the Armistice. But there is good reason to believe that the disease had long been endemic in parts of Poland and that it has persisted there ever since, although it was regarded as a form of malaria or influenza by the country doctors who alone were familiar with it. References to a *febris quintana* appear in the Russian literature a century ago, and the Moldavia fever described by Dehuo as occurring during the Russo-Turkish War in 1877 was almost certainly trench fever. Franke (1917) stated that he was familiar with the disease at Lemberg in Galicia before the last war, but that it was thought to be a form of influenza. The first German reference to trench fever was made by His in February 1916, he described it as *febris Wolhynica*, having observed it among German soldiers in the Wolhynia, a province of Poland just to the north of Moldavia and Galicia. The short form was subsequently known in Germany as "gaiter fever" and the long form as "five-day fever," corresponding with the earlier *febris quintana* in Poland. Trench fever was probably conveyed early in 1915 by lice-infested German soldiers from the eastern to the western front, where it soon spread to the British and French armies in France and Flanders. Its specific nature was recognised by Graham and subsequently by Hunt and Rankin and by McNee, Renshaw and Brunt a year earlier than the first observations recorded by the Germans and the French, and two years earlier than those of the Italians.

The clean clothes provided for all soldiers admitted into British hospitals prevented the conveyance of the infection to their homes when they went on leave, so that it never reached the civilian population in England. After the Armistice the small army still remaining on the Continent lived under such good hygienic conditions that lice infestation, and with it trench fever, quickly disappeared, and no further cases developed after the first weeks of 1919. Since that time it has never reappeared in the British army.

The low incidence of lice infestation in the Army in France in 1939 and 1940 would have prevented its development even if the disease had become endemic there but it died out after the last war as quickly in all parts of Western Europe as in the British army. As it had never occurred in England the herding of lice-infested people in air raid shelters did not result in the appearance of the disease as it certainly would have done in epidemic form had it been endemic in this country.

In 1922 the extracellular Rickettsia, *R. quintana* which is believed to be the causal organism appeared among the stock of 350 000 lice maintained for experimental work on typhus fever in Weigl's Pathological Institute in Lemberg where it caused an outbreak of fever among the fifty people employed as feeders for the lice. The infection was named Weigl's disease but it was clinically indistinguishable from the short form of trench fever. A similar event occurred in the same laboratory in 1938 (Herzl). It seems likely that the laboratory stock of lice became contaminated by the infected blood of feeders, who had contracted the infection in Lemberg where Herzl during the second epidemic actually isolated the organism in two patients unconnected with the Institute who were believed to be suffering from influenza.

If Weigl's disease is actually trench fever and if it has really continued to be endemic in Poland in the period between the two wars, it was certain to reappear in the lice-infested Polish, German and Russian armies. A German announcement in December 1941 explained that typhus-bearing lice had become more numerous and dangerous since winter began because of the difficulty of keeping clean and because the thicker clothing than worn harboured more lice and official Russian reports describe the almost universal lousiness of German prisoners. At least four papers appeared in German medical journals in 1941 and 1942 describing epidemics of trench fever in one of which it was stated that it was prevalent in Poland, Russia, Rumania, Italy, France and Belgium (Arneth, Jacobi, Kerger, Margert). It is of the utmost importance to keep soldiers free from lice infestation now that British and American armies are invading the Continent, and when they come into close contact with the Russian army not only in order to prevent typhus but also to prevent the much less serious, but very incapacitating trench fever.

Trench fever first appeared in the British army in France in the

early summer of 1915 It was at first observed only among officers and men living near the trenches and in the personnel of hospitals It was for this reason that the name "trench fever" was adopted, though actual residence in the trenches themselves was certainly not an essential factor, and subsequently cases occurred farther from the front, and even in very rare instances among orderlies in English hospitals, who had never been abroad but were attending patients from France Thousands of cases of the first type occurred among the troops in France and Flanders between the end of April and October 1915. It was comparatively rare in the following winter, but increased again in the spring of 1916 There are no available statistics as to the total incidence of trench fever, as the name was not sanctioned officially until 1917 Even at the end of the war more cases were labelled P U O (pyrexia of unknown origin) than trench fever, and a great many were also diagnosed as myalgia, rheumatism, debility and D A.H. It was estimated, however, that about 15 per cent of the total admissions for all forms of sickness into the clearing stations in France during 1917 were for trench fever (Soltau) As the average weekly evacuation for sickness (excluding wounds) from armies to base in France was 0.6 per cent of the strength, about 45,000 out of an army of a million would be lost each year by evacuation to the base Of these 90 per cent were off duty for at least three months

Trench fever did not occur in Gallipoli It appeared in Salonica in December 1915, and in Mesopotamia in May 1916 The second type, which was rare in France and Flanders until November 1915, when it became more common than the first, was the first to occur in Salonica, but it was not until we had been observing cases for three months that we read the first descriptions of trench fever from France and recognised that this form of P U O in Salonica was the same disease With the exception of one man who contracted the disease whilst in hospital for another disease, all of the cases observed in Salonica up to March 1916 belonged to two divisions which had been several months in France None had been in Gallipoli or Serbia The short form of trench fever had occurred in several of the affected units whilst they were in France It seems certain that these divisions brought the infection with them In April groups of cases of both forms of trench fever appeared in units belonging to other divisions, which had been free from the disease since their arrival at the end of 1915 The

infection in the later cases was probably conveyed by men coming in drafts from France.

### Pathology

From the first all investigators agreed that trench fever was not an aberrant form of some other condition such as paratyphoid fever which it may closely simulate during the first pyrexial period, true relapsing fever, which it resembles in so far as the fever is of a characteristic relapsing type, and malaria which is sometimes simulated by the shorter and sharper pyrexial attacks.

Reports were published from time to time in which various organisms were said to have been isolated from the blood in cases of trench fever but none of these observations was ever confirmed.

McNee and Renshaw found that trench fever could be transmitted to healthy soldiers by the intramuscular and intravenous injection of the blood of men suffering from the disease. This was confirmed two years later by the American Trench Fever Commission, which succeeded in transmitting the disease through three generations of men. The Commission found that the blood plasma contains the infective agent which is capable of passing through a Pasteur Chamberland filter with 740 mm. of mercury vacuum, and is destroyed by exposure to a temperature of 70° C for thirty minutes but not at 55° C though the latter is sufficient to kill the louse and its ova.

There is no nasal, pharyngeal or bronchial catarrh, and gastrointestinal symptoms are uncommon. It is probable therefore that the disease is not conveyed by the respiratory secretæ or by the feces, but through the intermediation of some insect. The occurrence of the long form of the disease during the winter months shows that the infection could be conveyed in the absence of mosquitoes and other flying insects. Though mosquitoes were found in France throughout the winter there were certainly none in Salonica. Fleas were scarce in both countries, and the men themselves rarely complained of them. Almost all patients with trench fever admitted that they were lice infested up to the time of their entry into hospital, so that it appeared possible from the first that the disease might be conveyed by lice. A hospital orderly who had been free from lice since his arrival in Salonica, had to carry the kit of a number of new patients suffering from trench fever on May 2nd 1916. The clothes were swarming with lice, and the same evening he found some in his own clothes. He got rid of them in the course

of a few days, and on May 20th an attack of trench fever began. He was not employed in the wards, he never came in contact with any patients suffering from the disease, and he was the first case of trench fever in the personnel of the hospital to which he was attached.

The incidence of trench fever was least in the cleanest battalions and the cleanest companies and platoons of battalions, and it was least in the divisions which had the best facilities for bathing. In some units a successful campaign against lice was immediately followed by a great diminution in the incidence of trench fever, and the campaign against lice in the whole Salonica Army in the spring and early summer of 1916 was followed by the almost complete disappearance of the disease. These observations led me to the conclusion that the disease was spread by lice. This was confirmed in April 1916, when Captain A. L. Urquhart developed the short form of trench fever after allowing the lice from a patient in Salonica with this form of the disease to bite him, and the same result was obtained in 1917 by Davies and Weldon in one out of two attempts. In 1918 the work of the American Trench Fever Committee proved conclusively that the disease is transmitted by lice. This was at first believed to be mainly by bites, but it was later shown that infection was conveyed by contamination of scratches with crushed lice or their excreta. Rubbing crushed lice or their excreta after the lice had fed on trench fever patients into the scarified skin of healthy men produced trench fever after a latent period, which averaged eight days. Lice become infective between eight and twelve days after feeding on trench fever patients and remain so for a fortnight. This suggests that the infective organism multiplies or completes a life-cycle within the louse. Lice do not infect their own offspring. The excreta of infected lice remain virulent for sixteen days when dried at the room temperature and after exposure to direct sunlight or to a temperature of  $56^{\circ}\text{C}$ , which is sufficient to kill the lice and their eggs, but the infective agent is destroyed by exposure to  $80^{\circ}\text{C}$  for ten minutes.

It is now believed that trench fever is caused by a variety of *Rickettsia*, a well-defined group of minute living organisms, the position of which in relation to bacteria and viruses is still uncertain. Whereas *R. prowazeki*, the organism of typhus fever, invades the epithelial cells of the midgut of the lice in which it lives, *R. quintana*, the supposed cause of trench fever, develops only in the lumen of

the louse a gut and never becomes intracellular. It multiplies for six to ten days within the gut and is then excreted in great numbers in the faeces.

Cold, wet and fatigue appear to be exciting causes in a man who has become infected, but has so far had no symptoms. Thus Hay noticed that almost all cases in his regiment in Salonica began two or three days after they had been wet through. Statistics of the incidence of the fever in a division in France between April 1915 and February 1916 showed that it was always highest during a period of stress (Muir). Thus it was much greater during periods of great activity in the trenches than after withdrawal into rest areas. This was not due simply to the removal from the trenches, as very strenuous training of a division whilst out of the trenches was followed by a rapid increase in the number of cases observed. Possibly however exposure to cold and wet act indirectly by inducing men to share blankets and sleep closer together so that infected lice pass from one to another with greater ease (Grievason). Over fatigue may also act indirectly as official methods of destroying lice are likely to be postponed in times of stress, and the men themselves are too tired to give up the necessary hour a day to destroy their own lice.

In many instances a group of men sleeping in the same tent or dug-out were infected. Grievason found that all but a very few of the numerous cases occurring in his battalion could be traced to small groups of men sleeping together. Some men appeared to be carriers, who did not lose the infection completely for several months, but had recurrences from time to time during each of which they infected an additional number of men. A sergeant, who had been in good health whilst in France between December 1914 and November 1915 developed the periodic form of trench fever early in December 1915 directly after he left France for Salonica. In the following four months he was in hospital five times for a week or more, though he was perfectly well in the intervals. Every time he returned to his unit he became lice infested again, and he appeared to infect most of the men with whom he came in contact, about forty men of his company, including six sergeants, having been taken ill with trench fever between January and March. One of the sergeants had wrestled with him, another had danced with him, a corporal slept next to him, and a private sat next to him for some lectures.

### Incubation Period.

Experimental infection with louse excreta showed that the incubation period was generally between seven and nine days, but it was prolonged to sixteen days when the doses were very minute (Byam). The latter period corresponds much better than the former with clinical experience, and in the experimental infection with living lice observed by Davies and Weldon the incubation period was sixteen days. As a result of observations in Salonica on cases arising in hospital in patients admitted for some other condition I came to the conclusion that the incubation period was between fifteen and twenty-five days. In the case of the hospital orderly already described it was probably eighteen days. Hunt and McNee in France concluded that it was between fourteen and twenty-four days, and Grieveson that it was about fourteen days.

The following four cases are typical of those which led to my estimate of the incubation period.

(1) Sergeant B, R E, was admitted for rhinitis on December 27th, 1915, into a ward in which there were at the time two patients suffering from the periodic form of trench fever, no other cases of which had been observed in his unit. On January 1st, 1916, he was moved into another ward, in which there were no such cases. On the evening of January 24th when convalescent from the rhinitis, which had been accompanied by no pyrexia or other symptoms, he suddenly became ill, his temperature at 6 p m was 104°. This proved to be the first pyrexial period of a typical attack of the periodic form of trench fever. The infection was probably contracted from the other cases in the ward between December 27th and January 1st, between twenty-three and twenty-seven days before the onset of symptoms.

(2) Private W went to France at the beginning of the war with the 1st — Regiment. He was wounded in January 1915, and was in England until the end of 1915, when he came to Salonica, joining the 2nd — Regiment, which had come there from France in November, on January 13th, 1916. A few days after he arrived he became lice infested. On January 21st he went to a field ambulance and then to a casualty clearing station with a hydrocele, he was transferred to a stationary hospital on February 6th. On February 12th his temperature rose and a typical attack of the periodic form of trench fever began. His clothes were disinfected when he entered the casualty clearing station, and he had no more trouble with lice after his admission there. It is probable that he contracted the disease whilst with his regiment, i.e., between twenty-four and sixteen days before the onset. As he was not lice infested until he had been with his regiment some days, the period was probably about three weeks.

(3) An officer was admitted under my care for "shell-shock." He

began to develop a typical attack of trench fever on October 27th. A brother officer who shared a dug-out with him, had returned to the front after an attack of trench fever about September 20th. The patient was much troubled with lice and only became free from them when he changed his clothes at a clearing station on October 3rd. He was probably infected by his brother officer in which case the incubation period must have been at least twenty four days.

(4) A fourth patient was admitted into hospital for quinsy. He was in a ward, in which there were no other cases of trench fever but he developed the disease fourteen days after admission. He had probably contracted it whilst still with his regiment, in which at least one case had already occurred, so that the incubation period was over a fortnight.

### Symptoms

Trench fever generally begins suddenly without premonitory symptoms, but it is occasionally preceded by a feeling of malaise for a day or two. The earliest symptom is severe headache, especially frontal and behind the eyes, and this is rapidly followed by pain in the lower part of the back and on the second or third day in the legs. The onset is sometimes extremely abrupt the patient suddenly feels giddy his legs give way under him and he shivers. He may be very short of breath and occasionally complains of pain in his left side. He has to fall out if on parade or marching, and has often great difficulty in returning to camp without assistance. Pain in the neck may occur it is occasionally accompanied by stiffness and may be sufficiently severe to suggest the presence of meningitis, which cannot be excluded without lumbar puncture. The patient generally shivers at the onset but there is never a definite rigor. He is sometimes flushed and often sweats profusely. The bowels are regular or constipated, and there is no nasal or bronchial catarrh. The appetite is lost, the tongue is moist and often slightly furred, and occasionally mild pharyngitis is present. Herpes labialis occurred in a few cases, especially in the short form of the disease. There is generally no rash, but Drummond observed a characteristic rash in between 30 and 50 per cent. of his cases of trench fever. It consisted of small rosy spots which do not project from the surface of the skin and are effaced by pressure. They appear in crops and occur chiefly on the chest and abdomen. They are rather redder than typical entero spots, have a less definite margin and do not project. They last for twelve to thirty hours instead of two or three days, and in relapsing cases usually appear a few hours before the fever. A

similar observation was made early in 1916 by McGavin in Salonica, but I saw the rash so rarely myself that I did not regard it as a symptom of the disease.

Drummond also described ocular symptoms, consisting of conjunctivitis, lateral nystagmus, generally persisting for three or four weeks, and in rare cases misty vision, sometimes associated with pain in the eyes

In a few cases, in which constipation is generally present, there is some abdominal pain with slight distension and tenderness, and there may be nausea and vomiting at the onset. Four out of my first fifty cases of the periodic type were sent to hospital diagnosed as appendicitis; in one a normal appendix was removed, and a second would have been operated upon had he not refused. The abdominal symptoms rapidly disappeared, and in the relapses they were less prominent than the other symptoms.

When the pain in the legs is severe, there may be some cutaneous hyperæsthesia over the shins. The shins are always tender, even if the patient complains of no pain in the legs, but tenderness appears to be most marked in groups of cases and at certain times. It was not observed in the earliest cases either in France or Salonica. The pressure of puttees may be enough to cause pain, and the patient often removes them in order to relieve his discomfort. In several cases the periosteum of the tibia seemed to me to be rough and thickened, and pitted slightly on pressure, although no pitting of the subcutaneous tissue was present. The tenderness is most marked over the lower half of the shins and may be very severe, a comparatively slight pressure causing the patient to cry out, and the pain produced may last for hours. A less degree of tenderness is often present in the tendons behind the knee, and occasionally in the ligamentum patellæ and along the course of the femur. In a few cases there was tenderness of the ulna on both sides. There is little or no tenderness of the calves or other muscles. The knee and ankle jerks are normal, and there is no evidence of neuritis, though a considerable degree of muscular atrophy may occur.

In the first attack of pyrexia the spleen is sometimes palpable. Although this was certainly the case in Salonica, Herringham, Hunt and McNee never found any splenic enlargement in the cases they observed in France in 1915, on the other hand, the committee appointed in 1917 to investigate trench fever reported that in 35 out of 91 cases seen in July, August and September 1917, the

spleen was palpable, and Boudin observed slight enlargement in the eight cases he saw in French soldiers in 1916.

Leucocytosis is often, but not always present during each febrile period. In many cases there was a relative increase in the large mononuclear cells. The percentage of hæmoglobin is generally about 80 though the number of red corpuscles is undiminished.

In the short form of trench fever the temperature rises rapidly to between  $102^{\circ}$  and  $104^{\circ}$  but the pulse rate is only slightly increased. On the third or fourth day the temperature suddenly falls—generally to normal or subnormal, but there is no corresponding improvement in the symptoms. After an interval of a few hours it rises again, and then after another two to five days



FIG. 18.—Short form of trench fever

it falls to normal on this occasion there is immediate relief to all of the symptoms (Fig 18). In some cases the remission on the third or fourth day does not occur the temperature remaining raised for about a week. There is often a single relapse after an interval varying from a few hours to ten days but generally less than four days the temperature rises to  $100^{\circ}$  or  $101^{\circ}$  for 24 or 48 hours, during which the symptoms return with diminished severity (Fig. 19). The patient is generally fit for duty almost immediately after the temperature falls again. Many cases were kept under observation by Hunt and McNee for weeks or months after the fall of temperature without any return of fever or other symptoms so that there could be no question of additional relapses occurring after the patient had been discharged from hospital.

In the *long* or *periodic type of trench fever* the temperature rises to between  $101^{\circ}$  and  $104^{\circ}$  on the first evening. The initial attack is variable in duration, the temperature may be normal the first morning, high in the evening, normal the second morning, and rather less high the second evening than the first, after which it remains down. In other cases the first attack may last as long as four or five days, the temperature being always lower in the morning than the preceding and following evening, the highest temperature being reached on the second or third day, in one case it reached  $105.8^{\circ}$  on the third evening, though it was normal the previous and following mornings. The pulse is generally accelerated

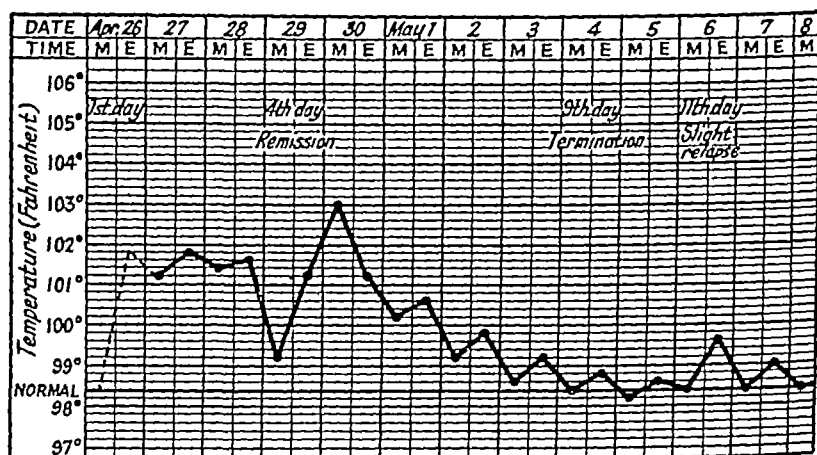


FIG 19—Short form of trench fever

in proportion with the temperature, but at first it may be considerably faster. With the fall of temperature at the end of the initial attack all the symptoms disappear and the patient is often sent back to duty.

After being well for two to ten days he complains of a return of headache and pain in the legs which culminate at night, the temperature rises in the evening to a point which is generally a little lower than the highest temperature in the first attack. The temperature falls to normal or nearly normal the next morning, and either remains down or rises to a less extent the second evening, thereafter to remain normal. The general symptoms are much less severe than in the first attack, and the acceleration of the pulse is less marked, but the pain in the legs and tenderness of the shins

are generally greater, and they may not disappear completely in the interval between the second and third attacks, though the headache, which generally remains the most prominent symptom during the attack, is never present in the apyrexial periods. The pain in the legs is sometimes extreme and may prevent sleep in other cases it is comparatively slight, and the patient looks and feels remarkably well considering that he has a temperature of  $101^{\circ}$  or more.

Recurrences follow periodically, the maximum temperature being always reached in the evening (Fig. 20). The intervals between the attacks are fairly constant in each case, but vary in different cases between four and eight days, five being the most common. Each succeeding attack is generally milder than its predecessor and the temperature is rather lower but in severe cases the patient feels weaker in the later intervals, and the pain and tenderness of the legs are more persistent. The later attacks may be of such short duration that the rise in temperature is not recorded at all if it is only taken twice a day (Fig. 21). On the afternoon and the evening of the day on which the attack is expected the temperature should therefore be taken every two hours, especially if there is any pain in the head or legs, as in most cases the patient knows from his sensations that there is going to be a relapse, even before the temperature rises. The temperature is sometimes raised only for three or four hours in one case, for example, the morning temperature was  $98^{\circ}$ , at 5.30 p.m. it was  $99^{\circ}$  at 6.30 p.m.  $100^{\circ}$  and at 8 p.m.  $101^{\circ}$  at 9 and 10 p.m. and at 8 a.m. the following morning it was  $98.4$ . In another it was  $97.6^{\circ}$  at 5 p.m. though the patient had had a headache since the morning, but  $101.2^{\circ}$  at 8 p.m.,  $102.4^{\circ}$  at 10 p.m.  $101.4^{\circ}$  at 2 a.m.,  $100.2^{\circ}$  at 6 a.m., and  $98^{\circ}$  at 8 a.m., so the morning and evening chart showed no rise, as the temperature in the ward was taken at 8 a.m. and 5 p.m. This liability for the rise in temperature to escape recognition accounts for the fact that it may appear from the chart that an attack has been missed, the interval between two of the later attacks being double that between the earlier ones. A headache may have been felt and a rise in the pulse rate recorded half way between the attacks. I saw a case in which the third relapse was of exceptional duration and severity this may have been due to a relapse having been really missed, as the apyrexial period which preceded it was of double length.



Occasionally the temperature remains raised for three or even four days in each attack, the evening temperature being always higher than the morning temperature, which may be normal on the first and last days the highest point is generally reached the second evening.

### Diagnosis

The diagnosis can be made with certainty from a study of the temperature chart, but the association of pyrexia with tender shins is very suggestive of trench fever already in the first attack. Painful and tender shins were, however occasionally observed in the Salonica Army in the apparent absence of fever and the unsatisfactory name of trench shin was sometimes used to describe such cases. It is, however not improbable that slight initial pyrexia whilst the patient was still at duty escaped notice. Some of the cases regarded as examples of the short form of trench fever are really periodic cases, as there is no doubt that the later bouts of pyrexia are often missed owing to the short time they last, the patient having meanwhile gone back to duty or if in hospital the evening temperature may have been taken at 5 p.m., although the rise only began at 7 p.m. or later. Several medical officers, who were very familiar with the early stages of the disease, recognised the periodic

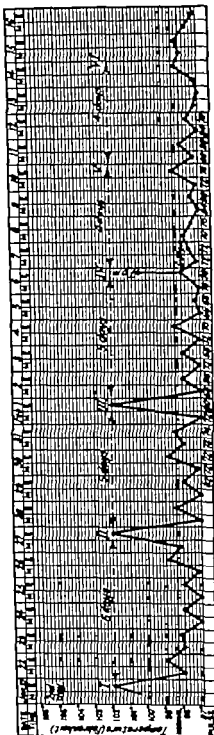


FIG. 21.—Periodic form of trench fever; the fourth rise of temperature occurred only after the evening temperature had been recorded.

rise of temperature after their attention had been specially drawn to its occurrence, as their patients had returned to duty after the first or second attack and had not "gone sick" for the later and comparatively slight recurrences

The majority of cases were at first diagnosed as *influenza*, though it was generally recognised that they were not identical with the familiar forms of that disease. Thus there was never any nasal or bronchial catarrh, the patient rarely appeared or felt seriously ill, except sometimes during the first two days of the first attack, and respiratory complications never occurred. The periodic return of pain and pyrexia and the pain and tenderness of the shins are quite characteristic and prevent confusion with *influenza* except at the onset.

The diagnosis from *dengue fever* may be very difficult, especially in tropical and subtropical coastal and inland river areas, where the vector of the latter, a mosquito, *Aedes ægypti*, abounds. The onset of dengue is sudden; the temperature rises rapidly to 102° to 103°, dropping by crisis three or four days later, but often rising again for two or three days after a short interval. Severe back-ache and joint pains occur, and a rash, described as half way between that of measles and scarlet fever, develops during the second pyrexial period. It is possible that the group of cases of trench fever recorded by Drummond in which a rash and conjunctivitis were present were really cases of dengue. Dengue is caused by a virus which undergoes a course of development in the body of *Aedes ægypti*, and the diagnosis can be ruled out in areas where this mosquito is not found.

The possibility of *malaria* must always be considered, and a blood film should be examined for the malarial parasite before making a definite diagnosis in cases of doubt, especially if the patient has previously had malaria, and in countries where it is prevalent, as was the case during the summer in Salonica. The longer intervals between the attacks, their invariable occurrence in the evening instead of at various times of the day, the absence of true rigors and the failure of quinine to modify the course of the illness are distinguishing features of trench fever. Several old soldiers at first thought that they were suffering from malaria, but they subsequently realised that the disease must be different, as they never before had had severe pain and tenderness in their shins.

A few cases of true spirochætal *relapsing fever* occurred in British

as well as Indian troops at Gallipoli. The disease was actually first described by Hippocrates in the Greek island of Thasos, and it has been endemic in the neighbourhood of Salonica since it was introduced during the Balkan Wars. It was common in the Serbian Army in 1915 and a few cases occurred among British and French soldiers in Salonica. There was a formidable epidemic among the Egyptian labour corps in April, 1916. 200 British troops in Egypt were attacked at the same time. Between May and August 1941 68 cases were admitted into an Australian general hospital in Tobruk. Two-thirds had lived in caves or old dug-outs previously occupied by Italians (Cooper 1942). The disease was remarkably mild, the mortality being only about 2 per cent. The initial pyrexial period was generally longer than in trench fever, varying between five and seven days and the maximum temperature was often over 104. In a large majority of cases only a single relapse lasting between three and five days, occurred after an interval of from eight to ten days. Headache was severe and pain was present in the muscles and the joints of the legs but not specially in the shins. Slight jaundice, slight enlargement of the liver and albuminuria were often present. The spirochete was found without difficulty in the blood during the pyrexial stage.

### Prognosis

There were no fatal cases of trench fever and the patient never appeared seriously ill, except occasionally for a very short time in the first pyrexial period.

The total duration of the periodic type of trench fever from the onset to the end of the last attack was generally between four and six weeks, but some cases appeared to abort, and in a few others attacks recurred for several months, the patient remaining quite well in the intervals. In most cases the patient rapidly recovered his strength after an attack and was generally fit for duty after the second period of pyrexia, though he might have to rest for a few hours when the later attacks occurred. Sometimes, however, great exhaustion followed and convalescence was slow.

About 85 per cent. of acute cases made a complete and rapid recovery. In about a third of the remaining cases disability lasted for six months or more. Men over the age of 35 and those who were already unfit when they developed infection were specially liable to pass into a chronic state of ill health.

Patients with trench fever who were kept in base hospitals too

long, and especially if they were transferred to hospitals in England, showed a great tendency to become chronic invalids, and large numbers were eventually discharged from the Army as cases of "neurasthenia" and "D A H." The former complained chiefly of exhaustion, irritability, headache, pains in the legs and back, the latter of breathlessness on exertion, palpitation, precordial pain, giddiness and fainting. I am convinced that the neurasthenia and D.A.H. were only to a minor degree a direct result of the toxæmia of trench fever and were mainly functional and preventable conditions. They developed most frequently in hospitals where over-conscientious medical officers were too much impressed with what they took to be a slight enlargement of the heart or an apical systolic murmur, and where an over-sympathetic nursing staff in English V.A.D. hospitals was too much impressed with complaints of fatigue and general unfitness. There was a remarkable difference in different hospitals both in France and England in the average stay in hospital of patients with trench fever coming from the same units, and my impression was that a long stay in hospital was the cause and not the result of a case becoming chronic. I do not think that there was ever any justification for invaliding a man from the service for trench fever, and no cases need be allowed to develop either neurasthenia or effort syndrome.

### Prophylaxis.

As the disease is conveyed by lice, which become infected by biting a patient during an attack, it should be possible to prevent the disease by keeping troops free from lice. All cases of trench fever should either be sent to hospital or isolated, and the patient's clothes and bedding should be specially disinfected, as well as that of all men who have recently slept near him. After the initial or the second attack a man is often able to return to duty. It is very important that he should be kept under observation, and if he again becomes lice-infested his clothes and bedding should again be disinfected. Men who are still having attacks or have recently recovered should sleep together, isolated from the other men in their unit, but there is no reason why they should not work with them.

A determined attempt to prevent the reappearance of trench fever in the present war is being made by the provision of sufficient baths, laundries and vermin destroyers and this has so far proved completely successful (July 1942). Between 1914 and 1918 men were frequently crowded in dug-outs and cellars and could change

their clothes or bathe at rare intervals with the result that lice-borne trench fever spread. Although measures to exterminate lice were energetically pursued from an early stage of the last war it was not until the middle of 1917 that equipment and facilities were sufficient to have any obvious effect in reducing the incidence of trench fever. \*The urine but not the faeces is infective there is rarely any sputum, but when obtainable it may also be infective (Strong *et al*) Precautions should therefore be taken to disinfect urine and sputum.

### Treatment

No treatment was found which prevented the periodic return of attacks. Aspirin was the most effective drug for the pains. Numerous local applications were tried for the painful shins some of them appeared to do good in certain cases, but the most frequently successful in my experience was a cold compress of saturated magnesium sulphate solution, which was first recommended by Captain D S Harvey

In a few cases in Salonica the periosteum was incised, but when this was done on one side only improvement occurred with equal rapidity on the opposite side. Even if the results had been more promising I should regard the operation as quite unjustifiable as the pain, however severe, always disappears spontaneously in the course of a few weeks and often quite rapidly

It is of great importance not to keep the patient in hospital longer than is absolutely necessary. He should be sent as soon as possible to a convalescent depot, where fresh air good food and progressive exercise quickly restore him to full capacity so that he is able to return to duty. If possible he should not be sent to hospital in England, as the longer he is in hospital the less likely he is to recover rapidly and completely

*Postscript.* Dr J G Greenfield has drawn my attention to the fact that there is one reported case of fatal trench fever. In Mrs. A. M. W. Stirling's biography of William de Morgan and his wife it is related that on December 26th, 1916 an Air Force officer who had returned from France the previous day came to see him. He had recently read *Joseph Vance* and had determined that his first visit in England should be to its author. Three days later de Morgan, who was then aged 78 felt strangely tired in the evening and before nightfall on the following day he was raving in the delirium of trench fever. This continued till he died seventeen days later and during all that time he believed he was a wounded soldier in a hospital in

France" His death is also ascribed to trench fever in the *Dictionary of National Biography* But the incubation period of trench fever is between fourteen and twenty-four days, never as short as three days, even when produced experimentally, and it is very unlikely that an Air Force officer would be wearing lice-infested clothes the day after his return to England Delirium was unknown in trench fever, and the acute symptoms and pyrexia never continued for more than nine or ten days, except in the long form, in which relapses, lasting for not more than a few hours, occurred about every fifth day. Mr de Morgan may have died from acute meningococcal fever, but he was certainly not a unique case of fatal trench fever

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## CHAPTER XIX

### LOUSE-BORNE TYPHUS FEVER

BY MELVILLE D. MACKENZIE, M.D.

Louse-borne typhus fever is an acute infectious disease lasting from twelve to sixteen days and characterised by a continued temperature, a generalised maculopapular rash which may become hæmorrhagic severe toxæmia and marked nervous manifestations. The disease is carried by lice and spreads with extreme rapidity especially through a badly nourished population. The causal organism is one of the group of *Rickettsia*—*R. prowazeki*.

Rickettsial diseases are widespread amongst animals and there are now few areas of the world where they have not been found. Louse-borne Rickettsial infection is however unique, in that, as far as is known, it is the only type that is primarily a disease of man and consequently the only one which occurs in epidemic form. All other Rickettsial conditions are, as far as man is concerned, endemic and localised being really accidental infections. It may seem strange that flea borne typhus does not spread in epidemic form like plague. This is probably due to two factors—in the first place the rat only carries the virus of the disease for a very short time and therefore relatively few fleas are infected secondly the disease is not fatal to the rat so that the fleas have no reason to drop off and seek other hosts.

In some parts of the world, particularly Mexico and Manchuria and also in Rumania, both the flea borne murine type and the louse-borne types exist side by side and appear largely to remain separate pathological entities. There is evidence, however that the ordinary flea borne murine typhus, if transmitted through lice, produces a disease both clinically and with immunological reactions indistinguishable from louse-borne typhus. From an epidemiological point of view this fact may be of very great importance particularly in Great Britain, where both the flea borne and tick borne forms of the disease have occurred within recent years.

Unfortunately it is impossible here to enter further into the extremely interesting facts now known regarding the transmutation of the *Rickettsia* viruses their varying virulence according to the

insect vector, and their relation to one another as shown by cross-immunisation and agglutination.

Typhus fever has until recently invariably been one of the most important diseases with which armies have had to contend, and a description of its ravages cover almost the whole history of military campaigns, particularly in Europe. Even with our knowledge to-day the impossibility in practice of keeping troops actually engaged in active operations free from lice makes the disease a very dangerous menace to soldiers fighting in an area where cases of typhus are occurring amongst the civilian population. Some idea of what typhus infection may mean is shown by the fact that in Russia alone during the period 1919 to 1922 the estimated number of cases was 10,000,000 with 4,000,000 deaths in a population of 120,000,000.

### **Predisposing Conditions.**

Epidemic typhus fever is classically associated with famine and overcrowding, but a third factor, which is perhaps of even greater importance, is the widespread movements of military or civilian populations, bringing non-immunes into a district where the disease is endemic or carrying the disease into a typhus-free region. Such movements may also introduce into an endemic region either a new strain of the disease or one of enhanced virulence. The first mode of infection was well demonstrated in the epidemic in North China in 1940, which was due to the introduction of masses of non-immunes with the army into areas in North China where the disease was endemic. The second and third methods occurred on the return of Polish prisoners of war to Poland from Siberia in 1919-22. These men, women and children had been heavily infected with typhus in Russia and passed into Poland at the rate of tens of thousands a day, going to regions in which the disease either was already endemic or did not exist previously. In both cases widespread epidemics resulted.

Apart from mass movements, a striking feature of epidemics is the amount of local movements of the population that they initiate. Once typhus is really established in a district, fear of contracting the disease, combined with terror of the appearance and acts of delirious patients, is soon widespread. Transport of food and fuel quickly breaks down, starvation threatens, the sick are abandoned, often in the roads, the houses are deserted, and the terrified population flees from the infected area into a neighbouring village or

another part of the town, carrying the disease with it. Too often the hospital staffs flee with the others.

Of the two other classical predisposing causes—famine and over crowding—I should certainly lay the greater stress on the former. Typhus spreads rapidly through a population where there is no overcrowding. On the other hand overcrowding alone is not sufficient to cause an epidemic in an endemic area, whereas famine certainly is. The importance of semi-starvation in spreading the disease is convincingly shown by the rapidity with which epidemic typhus disappears in a district once a supply of food becomes available and agricultural and economic reconstruction are effected. Overcrowding, inasmuch as it increases the number of lice and the facilities for the transfer of the insects, must obviously play an important part, but for the enhanced virulence required to maintain an epidemic in an endemic area I believe that a severe degree of under nourishment is necessary. Despite the fact that typhus is so closely related to malnutrition, it must not be forgotten that individuals who are strong and well nourished readily become infected and very frequently die.

#### Influence of Climate

Louse-borne typhus fever is a disease of cold countries and is unknown in tropical regions, though it may occur in the mountainous sectors. I have for example, seen it on the Equator in Bolivia at a height of 15 000 feet in winter and in Uganda the disease has occurred at an elevation of 5 400 feet. The distribution of louse-borne typhus may be said to cover all parts of Europe North and Central Asia, the line of the Andes in South America and localised sections of Africa, particularly in the north (see Fig 22). In considering the relations of louse-borne typhus to climatic conditions, it must be remembered that the behaviour of endemic typhus is different from that of the disease in epidemic form. In countries where the disease is endemic it shows a constant rhythm in its visitations. Year after year the curve starts in late November or December reaching an annual maximum in March or April, and continues until the end of June or July (Fig 23). It does not therefore actually coincide with the cold period of the year but is still widespread in the heat of May and June. Epidemics of typhus on the other hand, whether in an endemic area or amongst non-immunes, can occur at any time of the year. We have had an illustration of this fact in the recent epidemic in Spain, which started



in April and reached its maximum during some of the hottest months of the year (Fig 24) A further example was the occur

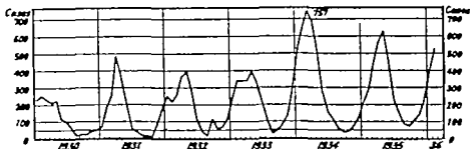


FIG. 23.—Typhus cases reported in Poland by four week periods from 1930 to 1936.

(From *League of Nations Epidemiological Report*, 1936.)

rence of typhus in Poland in 1910-20 (Fig 25) This was super imposed on an endemic focus, but continued in epidemic form throughout the summer, autumn and winter of 1919 returning to

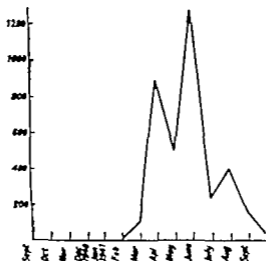


FIG. 24.—Typhus in Spain (Sept. 1940-41).

the normal endemic curve in 1920 with a maximum in the first six months of the year and an almost complete absence in the summer from the beginning of August onwards.

The method by which the disease in endemic areas is maintained between outbreaks is not definitely known. In an endemic typhus area one of the most striking features is its complete cessation after June or July. Repeated searching during the following few months fails to reveal any cases of the disease. Though undoubtedly "missed" cases may occur during the period between gross manifestations of the disease, I am very doubtful from the investigations I have been able to make whether these are sufficiently constant to maintain the virus from July to November or December each year.

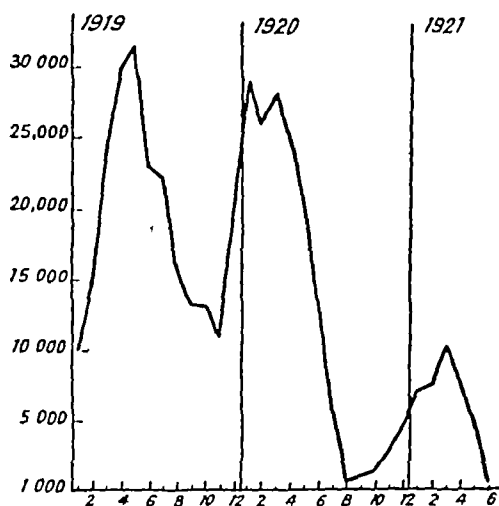


FIG 25 —Poland Typhus cases in 1919, 1920 and to July 1, 1921

There is of course the possibility that a proportion of the patients may remain as *Rickettsia* carriers. Cuca, Balteanu and Constantinesco (1935) have drawn attention to the difficulty of tracing infection from one case to another in non-epidemic periods, the failure to eradicate the disease from endemic areas by the isolation and delousing of definite cases, and the peculiar distribution of the disease and its comparative benignity in individuals born in endemic regions as compared with others, points which suggest the existence of mild, if not wholly inapparent, infections, which are yet capable of inducing immunity. An interesting difference between endemic and epidemic typhus relates to the virulence of the disease. In the endemic form the death-rate is generally low and fairly constant. In epidemics, although it is generally impossible to obtain accurate

figures the death rate is very variable, probably between 20 per cent. and 72 per cent. In giving such figures however it must be remembered that much depends on the age of the population and the hospital and treatment facilities. Moreover the figures are almost always based on hospital admissions, which do not include the mild cases amongst the children and younger members of the community

### Natural Immunity

The question of immunity in populations in endemic areas is of great interest. It is probably very rare for an individual to have a second attack of typhus. On the other hand, individuals coming into an endemic area as adults invariably get the disease more severely than adults of similar age in the endemic region. For instance in one of the endemic areas of China the death-rate amongst all Chinese adults admitted to a hospital under my supervision was 7.6 per cent. whereas that for Japanese who were new-comers to the region was 20.6 per cent. There were probably factors which rendered these figures not directly comparable but they bear out one's constant experience with the personnel of foreign relief units who develop the disease in endemic areas. One explanation referred to above is that a population in an endemic area may be partially immunised by a milder form of typhus, possibly murine which may pass unnoticed even by the individual. The virulence of typhus may be increased by passage through human beings, as there is no doubt the highest death rates appear in the last stages of an epidemic.

The severity of the disease varies greatly with the age of the patient. From extreme childhood the death rate progressively rises until after the age of fifty the disease is almost always fatal. I cannot recollect seeing anyone over fifty five, whether a native or foreigner who recovered in an epidemic area.

### Mode of Transmission

Epidemic typhus is carried as far as is known, only by the human louse of which there are three varieties—the head louse, the body louse and the pubic louse. The last is not recognised as a carrier of typhus probably not so much owing to its inability to carry the disease but to the fact that it lives on parts that are kept covered and consequently is not so easily transferred mechanically to another person. In addition, during its lifetime it moves very little if at all, from the immediate locality in which it was born.

A typical head louse has a different form from a typical body louse, but the great majority are not recognisable one from another. Moreover, head and body lice bred together produce offspring which are fertile. The head louse tends to live on the head and to lay its eggs in the hair, whilst the body louse lives generally on the body and lays its eggs mostly in the underclothing. Consequently, the head louse is not nearly so mobile as the body louse, which is easily brushed off by a passer-by or is shed in dressing and undressing. Nevertheless, for administrative purposes we must regard both the head and body louse as possible vectors of typhus fever.

The eggs of the human louse hatch about nine days after having been laid and the louse takes a further nine to ten days to reach maturity. The adult female lives about a month and lays eight to ten eggs a day, so that the total is in the region of three hundred. Lice live for about a week off the body at ordinary temperatures, and their eggs may hatch out over a period of about three weeks. Provided they are warm and have food at hand, they tend to remain where they were born. A louse rarely, if ever, voluntarily leaves an individual in cold weather unless he has a high temperature or is dead. Consequently the ordinary transference of lice from person to person is a purely mechanical process. Lice dislodged by rubbing shoulders in a crowd, shaken on to the pillow or sheets or during undressing on to the carpets and chairs of the bedroom, dropped as their host walks or sits down in shops, railway compartments, buses, and so on, are examples of how lice can be transferred mechanically and how typhus is spread. In a typhus area people quickly learn not to enter a crowd and take the greatest care not to brush against any passer-by. A louse finding itself in uncongenial surroundings, such as the outside of a garment on a cold day, rapidly moves in search of warmth and food. This is of great practical importance from an administrative point of view. It means that in a typhus area it is dangerous to wear either protective clothing or ordinary outer garments for more than two to three hours continuously.

Lice become infected with typhus by ingesting *Rickettsia* from the blood of persons during febrile periods of an attack of the disease, a period of about ten to fourteen days. They become infectious from the sixth day after feeding. In the gut of the louse the *Rickettsia* multiply and penetrate into the layers of the cells

of the gut. Those that enter the cells multiply rapidly so that the cells become distended and eventually burst. The louse dies about ten days after the rupture of the cells, but during this time the gut is packed with *Rickettsia* and large quantities are excreted with each dropping. These droppings when dry retain their infective power for a considerable time, and infection may occur from such dry faeces being rubbed into the skin, falling on the conjunctiva or possibly being inhaled into the lungs. There is as far as we know no regurgitation when a louse bites, as is the case in a plague-infected flea. The saliva, however, contains an irritant and man acquires the infection when he crushes the louse in scratching and rubs the contents of the gut on to his skin and into the puncture made by the insect. Whilst this is the ordinary method of infection, the danger of direct infection through the medium of the dried louse faeces cannot be dismissed. It is not known whether all the ordinary means of disinfection kill dried *Rickettsia*, but experience shows that if all the lice and eggs are destroyed in a community cases of typhus fever cease to occur.

### Pathology

The characteristic histological lesions are the so-called "typhus nodules." These may be very widespread but occur chiefly in the brain and in the skin. The lesion itself is a proliferation of the intima cells of the small blood vessels, the cells being crowded with *Rickettsial* bodies. As a result of the angitis the vessel walls break and small haemorrhages occur. Apart from these nodules there is nothing characteristic of typhus *post mortem* either to the naked eye or microscopically. There is, however, generalised congestion of the internal organs with marked myocardial degenerative changes. A combination of petechiae on the skin with congestion of the internal organs and an absence of any other obvious lesions is strongly suggestive, if not pathognomonic, of the death being due to typhus fever.

### Symptoms

*Incubation Period*—The commonest length for the incubation period is twelve to fourteen days, though periods varying from five to twenty days have been recorded. In practice it is difficult in epidemics to determine the time of the incubation, as the patient has often been exposed daily to the possibility of infection. The estimation is further complicated by the fact that he often feels "out of sorts" without being definitely ill for some days prior to

the rise of temperature For general administrative purposes the incubation period is taken as sixteen days

Typhus fever has been incomparably described by Murchison (1830-79) and one has only to work amongst typhus patients to realise his greatness as a clinician Here is his summary of a typical case

“ Occasionally it is preceded by one or more days of slight indisposition, characterised by lassitude, vertigo, slight headache and loss of appetite, but not such as to incapacitate the patient from following his ordinary employment With, or oftener without, these premonitory symptoms the patient is seized with slight rigors or chilliness, followed by lassitude and disinclination for exertion, frontal headache, pain in the back, pains like those from bruises in the limbs, especially in the thighs, loss of appetite and often, for a day or two, irregular chills and slight perspirations. For two or three days, although the temperature may be  $5^{\circ}$  or more above the normal standard, the patient complains of chilliness, and sits close to the fire

Occasionally there is nausea but rarely vomiting, the abdomen is free from pain, but there may be tenderness in the hepatic region, the bowels are constipated

The respirations are somewhat accelerated and sometimes there is a slight cough The face is flushed and dusky, the edges of the eyelids are tumefied, the conjunctivæ are injected, and the eyes water The expression at first betokens languor and weariness, but soon becomes dull, heavy and stupid

The sleep is disturbed by painful dreams and sudden starts, and after three or four nights there is talking in the sleep with slight delirium between sleeping and waking When awake the patient is still conscious, though perhaps somewhat confused in memory and intellect With all this there is early and rapidly increasing muscular prostration, the gait is tottering, the hand shakes, and there may be tremors of the tongue, soon there is an intolerable sensation of complete exhaustion so that about the third day the patient is compelled to keep his bed Between the fourth and seventh days, usually on the fourth or fifth, an eruption appears on the skin It is composed of numerous spots of irregular form, varying in diameter from three or four lines to a mere speck, which are either isolated, or grouped together in patches presenting a very irregular outline, and often closely resembling the eruption of measles At first these spots are of a dirty pink or florid colour and very slightly elevated above the skin and they

disappear upon pressure, but after the first or second day they usually become darker and more dingy they resemble reddish brown stains, are no longer elevated above the skin, and do not disappear on pressure. They have no defined margin, but merge insensibly into the colour of the surrounding skin. These spots usually come out first on the anterior fold of the axillæ and on the sides of the abdomen and thence they spread to the chest back, shoulders thighs and arms in some cases they are first seen on the backs of the hands they are most common on the trunk and arms and are very rarely observed on the neck or face. Along with these spots there are others which are paler and less distinct and which, from their apparent situation beneath the cuticle, have been designated subcuticular. When abundant, this subcuticular rash imparts to the skin a mottled or marbled aspect which contrasts with the darker more defined spots before described although sometimes the two appear to merge into one another. About the end of the first week, the headache ceases and delirium supervenes. The delirium varies in character. Occasionally it is at first acute followed by great collapse or the noisy condition passes into low muttering delirium. More commonly the delirium is never acute even at first. The countenance becomes more dusky while the prostration hourly increases. The tongue becomes dry, brown and rough along the centre and is tremulous sordes collect upon the teeth and lips. Gradually the eruption assumes a darker shade and about the eighth or tenth day true petechiæ of a purple or bluish tint appear in the centre of many of these spots. After three or four days the symptoms of nervous excitement are succeeded by more or less nervous depression and stupor. At first the stupor and delirium alternate the latter being most marked in the night time. The prostration is extreme the patient lies on his back, moaning, muttering incoherently or still and motionless. The expression is stupid and vacant. If spoken to loudly the patient opens his eyes and stares vacantly at those about him and when told to put out his tongue he opens his mouth and leaves it open until desired to close it. But all this time the mind is far from inactive the imagination conjures up the most frightful fancies to which implicit belief is attached, and of which a distinct recollection may remain after recovery. They who have passed through these mental sufferings can alone imagine their intensity. The teeth and lips are now covered with sordes the tongue is hard

and dry, dark brown or black, contracted into a ball, tremulous, and protruded with difficulty or not at all . The pulse is frequent, small, weak, and undulating and not unfrequently intermittent or scarcely perceptible . In this state the patient may continue for many hours or several days, with life trembling in the balance, until at last the stupor passes into profound and fatal coma . But on or about the fourteenth day there is often a more or less sudden amendment . The patient falls into a quiet sleep, which lasts for several hours and from which he awakes another man . At first he is bewildered and confused and wonders where he is but he recognises his attendants and friends and he is now conscious of his extreme debility . The pulse and temperature have fallen , the tongue is clean and moist at the edges , there is a desire for food . After two or three days the tongue becomes clean and moist all over, the appetite is ravenous, the pulse has fallen to its normal standard or is unusually slow, and the strength is rapidly regained ”

In the strenuous conditions under which extensive typhus epidemics occur the individual who is already weakened from malnutrition and exposure to cold often fails to notice the initial symptoms of the disease, so absorbed is he in the universal struggle for food, clothing, and warmth . Thus, though the ordinary history is that the patient had felt slightly indisposed one or two days previous to the onset of the temperature, the first sign noticed may be sudden mental confusion or a delusion in an apparently healthy individual . For instance, I recollect travelling on a locomotive from Kubyshev to Moscow with a British colleague, a healthy man of forty . It was winter, and we crouched in front of the engine furnace with the biting wind of the Steppe on our backs . My colleague remarked suddenly that he was an apple-green on one side and red on the other . This was the onset of a severe attack of typhus . Again, it is by no means uncommon for sudden homicidal mania or suicidal attempts to be the first symptom of illness noticed, and tragedies may occur through this cause . An interesting fact in connexion with these sudden onsets is the long distance patients delirious with typhus will walk in spite of the acute toxæmia of the disease, frequently under the impression that they are running away from lice . I have known delirious patients walk as much as ten miles over the open snow of the Steppe in the depths of a Siberian winter, clad only in a shirt, before they collapsed and were found to

be suffering from typhus fever. In the great majority of cases, however acute delirium is established only at the end of the first week, and a striking feature of the patient's condition then may be his clarity and normal mental state during the day at a time when he is wildly delirious at night. Two very constant symptoms at the onset are headache and bronchitis. The former may be frontal or occipital and its striking intensity may be a useful secondary diagnostic sign.

### Relapses and Second Attacks

Relapses in typhus, if they occur are exceedingly rare. Second attacks certainly occur but they are rare, and a single attack generally confers lifelong immunity. But the great difference in the virulence of the strains with the consequent variation of mortality in successive epidemics in the same country and the frequency with which patients in an endemic area state that they have previously had the disease perhaps as a child suggest that second attacks may be commoner than is generally supposed.

### Diagnosis

The clinical diagnosis of typhus fever is frequently exceedingly difficult. The conditions which favour its spread permit the occurrence of other diseases in epidemic form and, except in sporadic outbreaks in the vast majority of the cases I have seen the diagnosis was complicated by a double infection. Thus in Rumania relapsing fever and malignant malaria were widely epidemic at the same time as typhus in Russia relapsing fever malaria and smallpox in Poland relapsing fever and typhoid fever in China measles and a malignant form of scarlet fever in Bolivia influenza and pneumonia. The clinical picture presented by one, two or often three of these diseases in one being is obviously extremely complicated, and it is therefore of fundamental importance to be guided by the two clinical signs of the disease which are in my experience, the most reliable. These are the absence of the rash from the face and the fact that the rash does not appear in crops. It is said that very occasionally the typhus exanthem may occur about the base of the hair on the forehead and in the higher temporal region but for practical purposes its absence is the most important diagnostic sign we know. The absence of cropping has been repeatedly stressed since it was first noted by Stewart in 1840.

The date of appearance of the rash and its distribution on the trunk and limbs are too variable to be of great value in difficult

cases. Actually the exact day of the first appearance of the rash is difficult to determine, partly owing to the difficulty of saying exactly when the illness started, and partly because the rash may be extremely faint in its early stages. The commonest time is about the fifth and sixth days, but this is by no means constant. Peacock, for instance, in a series of 28 cases stated that the exanthem appeared in two on the third day, three on the fourth, five on the fifth, seven on the sixth, six on the seventh, two on the eighth, two on the ninth and one on the ninth or tenth. The presence of a rash in some form is, however, very constant, and in Murchison's cases an exanthem was noticed in 93.2 per cent of admissions to the London Fever Hospital. The rash is often faint and transient and may be absent in mild cases in children. My experience agrees with that of Browne Langrish, who as early as 1735 wrote, "Petechial spots and red efflorescence in large areas sometimes appear upon the skin. . . the brighter red they are of, so much the better sign. . . but when they appear of a purple brown or dusky or black colour they manifest a greater degree of putrefaction." In other words the deeper the colour of the rash, the greater its abundance, and the earlier it appears the more serious is the prognosis.

The character of delirium may be very characteristic. The delusions tend particularly to be of a terrifying type, with nightmare dreams, and may be associated with a fear of lice, which the patient imagines are consuming him, covering his pillow or dropping from the ceiling. Occupational delusions are common, and what one may describe as "dissociation" delusions are often characteristic in the later stages of the illness. Thus, the patient asks for his chin to be taken off for shaving, for his legs to be hung up at the foot of the bed or in the wardrobe, or he imagines he has left a leg lying about downstairs. I believe this type of delusion is of much value in diagnosis of cases in the second week of the disease.

The temperature is so often altered from the classical form by a second disease that, except in sporadic cases in non-endemic areas, it is of little guidance, at any rate in the earlier stages of the disease. On the other hand, errors of diagnosis have occurred through overlooking the fact that fever is invariably present in typhus fever, and cases of patients with mental illness and flea-bites have been thought to be typhus.

In sporadic cases, where the disease is commonly uncomplicated, in addition to the three classical symptoms of fever, nervous mani-

festations and rash, the most suggestive symptom in my experience is the general appearance of the patient. The dark and heavy flush of the face, the injection of the eyes, often with petechial hæmorrhages, and the dull stupefied look closely resemble the condition found in individuals in the later stages of an alcoholic debauch. Indeed, cases of alcoholism with vermin bites have been mistaken for typhus fever when the absence of temperature has been overlooked.

Apart from the clinical aspects of the disease during the febrile period, it is often important from an epidemiological point of view to detect cases which have recently had the disease but have recovered. At the beginning of convalescence the patient's mental condition is often far from normal. His mental processes are slowed down—he speaks very deliberately and cannot immediately reply to simple questions. This state may continue for some weeks during which an attack of mental confusion or even acute delirium may recur. A disconcerting symptom may be the sudden occurrence of transient delusions in a convalescent who is apparently absolutely normal mentally. A slight tremor of the hands persists for a long time and localized paralysis, particularly of a limb may occur during convalescence. Sudden syncope may supervene at any time in convalescence and in almost all cases great cardiac exhaustion is very manifest for some weeks after the illness. Localized gangrene, including cancrum oris is common in typhus and, though it often develops in the course of the illness it may only develop some time after the temperature falls. One or more of the above symptoms occurring in a typhus-infected area in an individual apparently weak, either from under nourishment or as the result of indefinite illness, may suggest that the person has had an attack of typhus. When it is possible to carry it out the Weil-Felix reaction is of course of value during convalescence in such cases.

*The Weil-Felix Reaction*—The diagnosis of typhus at the end of the first week can be confirmed by the Weil-Felix reaction. This depends on the fact that at the end of the first week or earlier the blood develops the power of agglutinating the so-called *Proteus X strama*. This reaction is specific for the typhus group of fevers and agglutination of *OX19* appears to be specific for louse-borne and flea-borne typhus. The *OX19* suspension issued by the Oxford Standards Laboratory should be used and the test carried out

macroscopically A strong reaction in a dilution of 1 : 80 or 1 : 100 may be regarded as positive when read by the naked eye, but in the later stages of the disease the titre usually rises to 1 : 1,000 or more. On the other hand, an increase in the agglutinating power of the serum, established by repeated examinations at intervals of two days, is significant, even at a titre of 1 : 50. From a purely epidemiological point of view two difficulties about the use of this reaction for diagnosis are that it occurs only relatively late in the disease—towards the end of the first week, and that under conditions of work in an epidemic the necessary *OX19* suspensions are not always available.

Of the differential diagnosis it is difficult to speak briefly, and in practice it is generally impossible definitely to diagnose the sporadic case of typhus fever before the rash appears on the fourth or fifth day. Until this time there are many febrile conditions which it is impossible to distinguish from it, as the Weil-Felix reaction is only present towards the end of the first week. Two common conditions which readily cause error in diagnosis in the earliest stage of the illness are the enteric fevers and the pyococcal infections of the central nervous system. A gradual rise of temperature, absence of the typhus facies, and the mental condition of the patient may be of assistance in excluding typhus in cases of the enteric group, but frequently it is impossible to reach a diagnosis until the results of cultures from the blood, faeces and urine and agglutination tests are available. In the case of the pyococcal infections early diagnosis on purely clinical ground is often impossible, and reliance has to be placed on the results of the examination of the cerebrospinal fluid and later on the rash and the Weil-Felix reaction. The existence of paralysis or a gradual rise of temperature are against a diagnosis of typhus. The absence of the rash from the face excludes measles and smallpox, though the prodromal rashes of the latter may closely resemble those of typhus in appearance and distribution. The fact that "cropping" does not occur in the exanthem of typhus helps in excluding the enteric group. Infections which have given rise to mistakes in diagnosis are acute febrile conditions, such as pneumonia and influenza, associated with vermin bites on the trunk. In the case of vermin bites the central puncture can readily be detected with a lens, and the lesion disappears on pressure except for the central dot. Malaria and relapsing fever can of course be excluded by blood films taken during the pyrexial stage. Moreover, malaria

reacts at once to quinine and relapsing fever to salvarsan. Double infections of these diseases with typhus frequently offer extreme difficulty in diagnosis.

### Treatment

Apart from the use of convalescent serum, the treatment of typhus is entirely symptomatic. Unfortunately there is no evidence that sulphonamides are of any value. The first step is the complete disinfection of the patient, a process which should be repeated ten days later in order to destroy any eggs that may have been missed and which have since hatched out. Sedatives and often physical restraint are required in the earlier stages of the disease. The nursing is of the greatest importance, particularly in relation to oral hygiene and to bedsores as the illness is almost invariably accompanied by faecal incontinence. The patient should be encouraged to drink as much water as possible. Increasing cardiac weakness may call for carefully considered treatment. The exhibition of such stimulants as alcohol may sometimes be of value in tiding a patient over a critical time but its value is much more limited than was formerly believed.

Complications are fortunately not common during the acute stage, but bronchitis is frequently present and calls for the ordinary therapeutic measures.

### Control of Typhus Fever

Anti typhus units consisting of doctors, nurses and subordinate medical auxiliaries should be organised and trained beforehand whenever cases of typhus may be anticipated. In view of the much lower death rate from typhus at early ages the personnel of these units should be as young as possible and in any case not more than forty years old. As an attack of the disease almost invariably confers lifelong immunity use should be made wherever possible of individuals who are known to have had typhus. It is desirable to recruit personnel as far as possible from doctors and nurses coming from areas where typhus is endemic, partly for the above reason and partly for their knowledge of the disease.

Too strong emphasis cannot be laid on the great danger to which personnel who work amongst typhus patients are exposed and on the need for careful personal supervision of their activities by a medical officer. The most dangerous parts of the work are the search for cases in houses, the work in the admission block of a typhus hospital, and the de-lousing of contacts of patients, their

clothing and bedding, but in all typhus work no unnecessary risk through slackness in taking the necessary precautions must be tolerated. Although we have no definite knowledge of any vaccine which certainly protects under field conditions, all workers should be offered the possible protection conferred by inoculation. The hair of all medical personnel dealing with typhus should be cut short and if possible shaved, including hair on the body. Protective clothing must invariably be worn. This can be made of any closely woven strong cotton fabric, and should be dark blue or khaki in colour in order to show up lice. The garment itself should be made in one piece, including a hood for the head, the legs being continued into the feet as in fishing waders. It is completely closed except at the back, round the face and at the wrists. The back opening is closed by a zip which, if properly fitted, gives most protection, it should be non-rusting. If zip fasteners are not available, the aperture down the back should be closed with tapes stitched on the outside so as to allow an overlap of at least eight inches. The hood is fitted with a tape running round the face aperture and tied below. Tapes and elastic are provided to draw the sleeves closely round the wrists. All seams in the garment must be made with two rows of stitching. Gloves and gumboots must also be worn. The gloves should be of rubber or cotton fabric and should come well up the forearm and grip closely over the sleeves. Lighter forms of gum-boots are the most suitable for ordinary work.

The garment described above has the practical advantage that even in extreme cold it often obviates the need for the use of furs. To be effective from this point of view, however, it must be made of cloth that is absolutely wind-proof and be loose fitting, and the wearer must be actively employed. For relatively sedentary work furs are necessary and must be worn underneath the protective clothing, a number of protective garments of extra size being provided for the purpose. The need for nurses to wear protective clothing depends on how far confidence can be placed on the efficacy of the disinfection on admission. If this can be relied upon completely, as in the case of first-class typhus hospitals under normal conditions, only ordinary nursing uniform need be worn in the wards. Under epidemic conditions, however, and working, as is often the case, with a largely untrained staff in the admission block, it is impossible to be certain that every louse is destroyed prior to admitting the patient to the ward, and consequently the

special garments described above should be worn constantly by the nurses. The garments must be invariably used by all who search for cases in houses or elsewhere, all engaged in disinfection work or working in the admission block of a typhus hospital.

It is clearly impracticable for all those engaged in general work in a typhus area to wear protective clothing but the risk of infection can be greatly minimised by having the hair short the body hair shaved frequent bathing and by invariably wearing a smooth cloth raincoat or rubber mackintosh coming down to below the knees in conjunction with long boots or puttees. Fur coats should be worn with the fur inside and the raincoat on top.

Such lice as may have been acquired by mechanical transference are on the outside of the coat. Some little time is required for them to make their way on to the wearer's skin and it is for this reason that it is most important to carry out disinfection of outer clothing at as short intervals as possible.

One of the greatest risks in typhus work arises when it is necessary to live and sleep in squalid peasants' houses, railway trains etc., in a heavily infected area. The chance of infection may however be greatly lessened by the use of a special sleeping bag. This can be of any closely woven cotton material and should be made in the form of a sack ten to twelve feet long and fitted at the open end with a zipper or circular tape which can be drawn tight and fastened up from the inside. Inset in an aperture over the face, are two thicknesses of muslin supported on a light metal frame. The whole sleeping bag can be boiled and gives protection from the attacks of all insects—bugs, fleas and lice.

Arrangements must also be made for the frequent bathing of personnel, where this is possible and for the regular disinfection of their garments and sleeping bags. The stores required include portable baths or showers, fuel for heating water soap hair clippers scissors nail brushes towels etc. in addition to as good rations as it is possible to obtain.

*Control of typhus fever in the field*—As no protective vaccine of proved efficacy in the field is known with the possible exception of Weigl's vaccine, the only means of controlling typhus fever is by the destruction of lice on both the body and clothing of all those infected who may have been in contact with a case. Lice and nits are killed by exposure of five minutes to a temperature of 55° C. In actual practice the temperature should be 80° C. and clothing

should be exposed for thirty minutes. The insects and their eggs are killed instantly by exposure to moist heat at a temperature of  $80^{\circ}\text{C}$  or over, but in practice a temperature of  $100^{\circ}\text{C}$  should be used. Bathing and de-lousing of clothing should be repeated at weekly intervals. It is obvious that the possibility of checking an epidemic depends upon how conscientiously this work is carried out, and careful study should be made beforehand, not only of the best means of de-lousing under local conditions, but also of the details of the safest technique by which the patient can be removed from his house, admitted to, and treated in hospital, so as to subject those doing it to a minimum risk of infection.

The destruction of lice must obviously be done simultaneously on the body and the clothing. Arrangements must therefore be made for the individual to be bathed and if necessary shaved whilst his clothing is being treated. In any area where typhus is epidemic, accompanied as such outbreaks almost invariably are by a state of chaos, it is impossible to kill all the lice on all individuals. Experience has shown, however, that a definite reduction in the general degree of infestation of the population is followed by a greatly lessened number of cases of typhus and relapsing fever.

The ordinary method of de-lousing which are available in an organised community are fully described in the various textbooks and military manuals and need not be described here. There are, however, several important special points in connection with de-lousing for typhus to which attention should be drawn. In the first place it is clear that as the disease can readily be carried by a single louse, disinfection of the patient, the "contacts," the ambulance, the premises and the personnel employed must be absolutely complete and must therefore be under the direct supervision of a medical officer. Not only must this officer assure himself beforehand that whatever disinfecting machinery it is proposed to use is effective, but during disinfection he must make certain that it is being correctly used. Secondly, rapidity of action is the keynote of typhus control in order to destroy infected lice before they become infectious, i.e. in six days after feeding. Thirdly, the method of de-lousing by "hand-picking" is to be strongly deprecated in typhus areas. It is impossible to do this without crushing a certain number of insects with the risk of auto-inoculation. Finally, whilst it may be desirable to utilise every method in dealing with typhus-infected lice, the greatest care must be taken that the use

of insecticide powders does not constitute a danger by creating a false confidence which leads to neglect of the well proved methods of protection and disinfection. Little is known of the action of these preparations on the *Rickettsia* themselves and their value lies chiefly in their deterrent properties against lice. The greatest caution should be exercised in placing any reliance upon them in anti typhus work.

It is essential to reserve an ambulance for typhus cases, and the vehicle which is chosen should be one which lends itself to complete cleansing of the interior and which offers no harbourage to insects. Every care must be taken to transport the patient from his bed to the ambulance in such a way that lice are not shaken off him. This is best done by completely enveloping him in a large sheet ten feet long, during the journey from the bedroom to the ambulance. It is not necessary here to enter into all the details of the disinfection of cases on arrival at hospital prior to admission to a ward, as these will suggest themselves in the light of the observations made above particularly with reference to the danger of the transference of the disease to personnel. There are, however three important points. Firstly every hospital intended for typhus cases should be provided beforehand with a properly equipped admission block and a disinfecting apparatus and should be staffed by young personnel trained in thorough disinfection. Secondly in addition to the destruction of the lice the greatest care should be taken to ensure that all nits are also dealt with. The blood of a typhus patient is highly infectious for the two weeks of illness and a nit which has been overlooked may hatch out, feed on the patient, and eventually cause the disease in a nurse. For this reason all patients should undergo a second disinfection ten days after they have been admitted to hospital. Thirdly it is unnecessary to use disinfectants in the bath given to the patient as it is impossible to utilise a sufficiently strong solution of an ordinary disinfectant to ensure the death of the insect in the relatively short time in which it is exposed, and at the same time not to damage the patient. The removal of the lice by bathing is mechanical, and ordinary soap possibly soft soap mixed with paraffin, is sufficient if conscientiously applied with a nail brush after the patient has been shaved.

Provided disinfection has been properly carried out, it is no more dangerous to nurse a case of typhus than it is to nurse a case of typhoid fever or of plague freed from fleas. It must, however

should be exposed for thirty minutes. The insects and their eggs are killed instantly by exposure to moist heat at a temperature of  $80^{\circ}\text{C}$  or over, but in practice a temperature of  $100^{\circ}\text{C}$  should be used. Bathing and de-lousing of clothing should be repeated at weekly intervals. It is obvious that the possibility of checking an epidemic depends upon how conscientiously this work is carried out, and careful study should be made beforehand, not only of the best means of de-lousing under local conditions, but also of the details of the safest technique by which the patient can be removed from his house, admitted to, and treated in hospital, so as to subject those doing it to a minimum risk of infection.

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informed of the existence and extent of the disease in their locality and if necessary a brief description of some of the important diagnostic signs should be circulated. The ease with which cases of typhus are missed is well shown in figures given by Davidson and Cruckshank. In the period 1901 to 1926 87 groups of cases occurred in Glasgow, and in 36 of these the diagnosis of the primary case was missed. The analysis of the diagnosis of the first case in each group was typhus 51, enteric fever 22, pneumonia 5 other diseases 9.

As typhus may masquerade under such varying clinical forms, all death certificates should be scrutinised by the Medical Officer of Health for suspicious deaths, and he should keep in constant touch with all doctors in his area engaged in general practice. In addition a close search for cases must be maintained by the Medical Officer of Health and his staff, particular regard being paid to cases of illness where a doctor is not in attendance. In view of the mildness of the disease amongst children, absentees from school must be closely investigated. Propaganda informing the public how the disease is carried, how lice can be killed, and outlining the disinfecting facilities available is of the greatest importance.

In the event of troops being moved into an area, where typhus is widely epidemic amongst the general population, it is of primary importance to do as much as is reasonably possible to control the disease amongst the civilians often in the absence of any civil authority. The commonest and most difficult case occurs when the disease is widespread over vast areas, such as the whole of Poland, Rumania and the Ukraine, amongst illiterate peasants with little or no organised health service. Under these conditions it is obvious that effective work is beyond the capacity of imported disinfecting plants, which, being limited both in number and by the time taken for each operation, could only deal with a minute fraction of the population. Moreover as rapidity is the key note in action against typhus, the delay entailed in obtaining and distributing disinfecting apparatus greatly reduces its practical value. Any method for the immediate control of epidemic typhus under the conditions which may be expected in many parts of Eastern Europe must be one that can be applied at once and consequently one which the population can carry out for themselves with the facilities available in the villages. The first step is to convey to the whole population the fact that typhus fever is carried by the human louse and by the human louse only. Many persons will already know this, probably

be remembered that the blood of typhus patients is highly infectious, and consequently great care must be taken in collecting blood samples and in nursing patients who are coughing blood. The excreta are, at any rate theoretically, infectious. The question of the possibility of droplet infection from the cough is undecided, and some authorities urge the necessity of wearing masks for nursing, whilst others do not consider there is any appreciable risk from this cause.

Typhus fever is the only disease which may be met with in Britain which we know may be dangerous during the incubation period. Throughout the twelve or fourteen days after infection, before the illness manifests itself, the patient constitutes a potential danger through the infected lice he may carry. A very large number of contacts may thus be created, so that once the disease is really epidemic in an area, it is useless to attempt to deal with more than the immediate (family) contacts, and efforts can be usefully directed only to the reduction of the louse population generally.

With regard to the disinfection of the premises, cyanide gas is probably the most satisfactory, though its action on the *Rickettsia* is uncertain. We have no evidence that sulphur or formalin, as applied in ordinary disinfection, has any lethal effect on lice or *Rickettsia* bodies. Armchairs, sofas, wardrobes, etc., are all likely to be infective in the bedroom of a typhus case, but as they cannot be readily disinfected in an ordinary disinfector, and as there could be danger in handling and transporting them, disinfection *in situ* is essential. Bedding and clothing of the patient and the immediate contacts should be thoroughly disinfected with steam. As lice, if unfed, die in ten days, the simplest method of de-lousing a house, including the furniture, is to shut it for a fortnight, sealing the doors and windows and burning a sulphur candle to prevent the inhabitants from re-entering it. As the disease is not transmitted through the egg, no danger is to be apprehended, even if these survive, provided the patient has been removed.

With regard to disinfection of bedding and clothing, though ideally a disinfecting station should be reserved for typhus work, in practice this is rarely possible. If an ordinary disinfecting station is used, it must be closed for all other purposes until all the typhus work is done and the station has been thoroughly cleansed. It is essential to be absolutely sure that the disinfecting machinery is in proper order and correctly used.

Immediately typhus occurs, all practitioners should be kept

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the majority, but it is nevertheless an essential preliminary to further activities to convey this information to as many people as possible in the affected area. The second step is to describe in an absolutely simple way the best method of getting rid of lice from the body and the clothing, giving exact details as to the shaving and bathing necessary, as well as to the disinfection of the clothing. In cases where facilities for radio reception exist in villages, these two preliminary steps can be carried out by wireless immediately. Otherwise the information may be conveyed by Government instructions to the mayor of the township or village or by Government posters, though for the illiterate these must be largely pictorial.

So great is the fear of typhus that a large proportion even of a peasant population will carry out any instructions given. Cutting the hair can obviously be done by all. With regard to bathing the chief difficulty is the supply of soap, and one of the earliest measures may be to arrange for its distribution to the villages in each area. It will be found that with little guidance arrangements can generally be made for bathing either in the public baths, commonly of the modified Russian type, with which most villages in Eastern Europe are furnished, or in the houses of individuals. In Eastern and much of Central Europe the village bathing place in rural areas is the local river or pond in summer, and in winter, except for baths of the Russian type, little if any bathing is ordinarily done. In villages where a Russian or some other form of public bath does not exist, it is necessary to explain in detail by means of wireless or Government instructions how to improvise a bath in one of the fresh-water butts with which every house is provided.

Cotton garments can be dealt with most conveniently by boiling. The most generally applicable method of destroying lice in woollen materials under conditions of epidemic typhus is by the application of heat by ironing. This has many advantages, it is simple in application, easy to explain to ignorant people; most households possess an iron, or where necessary irons can often be distributed, repeated applications are possible, and the work can be carried out at regular intervals by the woman of any household, who is ready to protect her family, independently of any outside arrangements. Perhaps the greatest value of ironing is, however, that it can be used in the disinfection of sheepskins and furs, which will not stand the application of steam or the dry heat temperature reached in an oven.

In many parts of Eastern Europe the Russian bath can be utilised. This is a wooden or brick hut in which a fire is made under heaps of stones, which are thus heated to a high temperature. Buckets of water are thrown on the stones the water immediately evaporating into clouds of steam. The population are first bathed and de-loused in the bath house, and then the amount of heat and steam is increased so as to deal with the bedding and clothing. Subsequently no further water is thrown on the stones and the heat of the hut is allowed to dry out the material.

In districts where such baths do not exist it is often possible for the peasants to build brick ovens. These can be made of local unburnt brick rapidly and in large numbers and heated with wood fuel. The material for disinfection is introduced and left for half an hour care being taken to hang the bedding and clothing so that provision is made for the complete circulation of hot air. A useful modification of the oven is an underground pit or excavation in the side of a bank, which can be heated to the requisite temperature by braziers placed within it the heat being retained by a curtain hung over the entrance.

In considering improvised disinfecting methods, a word of caution is necessary with regard to the use of Serbian barrels, sack, and other disinfectors which depend for their efficacy upon the use of current steam. Not only are they difficult to employ on a really large scale owing to their small individual capacity but the fact that the garments emerge wet is a very great disadvantage, especially in a country where all moisture freezes hard at once. In dealing with sporadic outbreaks in a climate which allows of the hot garments drying when shaken out Serbian barrels, sack and other current steam disinfectors may nevertheless be of great value.

None of these methods is, however applicable to the commonest wearing apparel in Northern Europe in winter namely sheepskins and furs. In winter in a cold country it is sufficient to leave the furs on the outside of the house in the open during the night for every louse to be destroyed. Whether the nits (eggs) survive or not depends upon the degree of cold, but in any case there is no evidence that these can transmit the disease. When the nights are not sufficiently cold the only possible way of dealing with sheepskins in the absence of imported chemical disinfectants such as naphthalene, is by ironing.

Finally mention should be made of two methods of louse destruction which, though still in the experimental stage, may eventually supersede many of the older methods of disinfection. The first of these is the utilisation of various powders lethal to lice, which are used either for dusting garments or for the impregnation of clothing. These powders are undoubtedly of the highest value, not only as disinfecting agents, but also as deterrents, and are being extensively used in the Allied Armies with increasing success. It must be remembered, however, in all louse control work, that it is one thing to deal with a disciplined group of men, where the regular use of the powders can be enforced, as contrasted with a civilian population, often illiterate and under no direct supervision. So far, no opportunity has arisen to utilise these insecticide powders on a wide scale amongst an ordinary civilian population.

A second new method of disinfection depends on the utilisation of methyl bromide. Clothing is placed in a special sack with an ampoule of methyl bromide. The sack is closed and the ampoule broken. At the end of half an hour the sack is opened, a period of ten minutes is allowed for ventilation and the operation is complete. Certain dangers in the use of methyl bromide call for precautions and necessitate carrying out disinfection work in the open air. From a practical point of view, however, the greatest drawback is that, owing to the temperature at which methyl bromide volatilises, the method is not practicable when the climatic temperature is low, i.e. in cold countries where typhus is most likely to occur. Experimental work is at present proceeding in connection with other chemicals similar in their action to methyl bromide but which volatilise at a lower temperature.

Those who wish to study more fully the clinical aspects of typhus fever are recommended to read *Continued Fevers* by Murchison, 1884, *Infectious Diseases* by Claude Ker, 1920, and *Clinical Practice in Infectious Diseases* by E. H. R. Harries and M. Mitman, 1940.

For the biology of the louse reference should be made to *The Louse* by Buxton and *Lice* by Smart.

## CHAPTER XX

### TYPHOID AND PARATYPHOID FEVERS

Before 1914 paratyphoid fever B was very rare in England compared with typhoid fever and it was unknown in India. Many British soldiers became infected with it in the early months of the war in part from the civil population close to the front, especially the Belgian refugees, and in part as a result of the reoccupation after the battle of the Marne of territory which had been fouled by paratyphoid carriers among the German invaders. It was later conveyed to all the theatres of the war by soldiers drafted from France. Paratyphoid A was well known in India, though it had rarely been recognised in Europe before the war. The majority of the earlier cases which occurred in France were among troops from India, but its high incidence in Gallipoli and its much greater frequency than paratyphoid B in Salonica showed that it must have been endemic, though hitherto unrecognised, in these areas. It is interesting to note that whereas paratyphoid B is probably very much more common in Great Britain now than before the last war whereas typhoid fever is much less common, paratyphoid A has never spread among the civilian population in England and is now almost unknown.

In the four years between 1914 and 1918 with an average strength of nearly two million men in the British Army there were only 20 149 cases of typhoid and paratyphoid fever with 1191 deaths, giving a case mortality of 5.4 per cent. About one-third of these occurred in France. The incidence there was highest in 1915 and 1916. In spite of the growth in size of the Army the number was halved in 1917 and in 1918 with a ration strength of two and a half million there were only 90 cases of typhoid 43 of paratyphoid A and 156 of paratyphoid B. The contrast between this record and the 57 684 cases with 8,022 deaths in the army with an average strength of 208,226 which fought in the South African War is remarkable.

The number of cases of paratyphoid B in France formed at first three-quarters but later only two-thirds of the total of A and B. The incidence of the disease steadily diminished after the first four months in spite of the increasing size of the army and in November

1916, there were only 30 cases of typhoid and 92 of paratyphoid fever in hospital in France—the lowest number since October 1914. Paratyphoid fever was considerably more prevalent in Gallipoli and Lemnos, where it was probably ten times as common as typhoid fever. According to statistics published by Martin and Upjohn, about 5,700 cases of enteric fever occurred among the 96,683 medical casualties, which occurred in the 300,000 men who fought in this area up to the middle of December 1915, and of these about 93 per cent. were paratyphoid and only 7 per cent. typhoid fever. Paratyphoid B was the prevalent type up to the end of October, when paratyphoid A became more common, and by December it had almost entirely replaced paratyphoid B. Paratyphoid A was also very much more common than paratyphoid B in Mesopotamia and in Salonica, where the total incidence was small. A few cases of paratyphoid C were recognised in these areas. The incidence of enteric fever in the Army in the Middle East in 1940 was 0.07 per thousand compared with 1.89 in the war of 1914–18. It has remained low since then, although it was common among Italian troops in Libya.

### **Ætiology.**

The primary source of infection with typhoid and paratyphoid fever is a patient suffering from the disease or a carrier who has had the disease, as the infecting organism may still be present in the faeces a year after the illness, water or food becoming contaminated by his excreta directly or more often indirectly by flies and dust. The sputum has been found to contain the organism in cases complicated by broncho-pneumonia, it must therefore be regarded as a possible source of infection for nurses, orderlies, and other patients in the ward.

### **Morbid Anatomy.**

There is nothing about the morbid anatomy of the paratyphoid fevers, which clearly distinguishes them from typhoid fever or from each other. There is perhaps a tendency both in paratyphoid fever A and B for the colon to be more widely involved compared with the small intestine than in typhoid fever, as ulcers are often present from the caecum to the splenic flexure, and I have even seen them in the rectum, but this is not a constant feature, for in two cases the small intestine was alone infected and in a third—a case of paratyphoid A—no ulcers were present in any part of the inflamed intestines. Carles (1916) refers to six similar cases recorded by

French observers, and to one in which the intestines were not even inflamed.

### Symptoms

The symptoms of typhoid and the paratyphoid fevers are so much alike that they are best described together. The incubation period is between three and twenty days. The onset is generally more or less acute. The patient always complains of headache, often of abdominal discomfort and sometimes of diarrhoea. The diarrhoea soon ceases, but the headache persists and pains in the back and limbs develop so that by the fourth day he generally feels too weak and ill to continue with his usual occupation. In some cases severe headache, abdominal pain with or without diarrhoea, and repeated shivering attacks develop so acutely that in a few hours the patient is obviously ill. Epistaxis, so slight that the patient generally does not mention it unless specially asked was a common early symptom of paratyphoid in France.

The headache may be sufficiently severe to prevent sleep the first few nights, after which it gradually improves. Diffuse abdominal discomfort is common. It rarely amounts to pain and generally lasts only two or three days, but in very acute cases it may be severe and accompanied by vomiting. The tongue is furred, and in severe cases it is dry and brown. If an entirely fluid diet is given it remains coated except at the tip and edges. I have, however, generally found that if the patient takes biscuits and other dry food, which entail chewing and promote the flow of saliva the tongue remains clean and moist throughout the illness. The slight diarrhoea, which occurs in more than half of the cases at the onset, is not severe, a small number of loose stools being passed for a few days, after which constipation is almost always present.

The abdomen may be normal in appearance but it is more often full. There is generally no tenderness apart from that of the spleen. The spleen is palpable in at least two-thirds of the cases and in my experience of paratyphoid fever it is found to be enlarged by percussion in all of the others. It is often much firmer in paratyphoid than in typhoid fever and may remain palpable for some days after the temperature has fallen to normal. It is generally tender in the early stages and may give rise to spontaneous pain, which is increased by deep inspiration and coughing and is occasionally so severe that the onset of pleurisy is suspected. Even when it is

impalpable, pain is produced by deep pressure under the outer part of the left costal margin. The liver is not enlarged. The gall-bladder is occasionally tender owing to the presence of cholecystitis, which may give rise to no other symptoms.

In addition to the headache and abdominal discomfort, the patient often complains of pains all over the body, especially in the back and limbs and sometimes in the joints, which are not, however, swollen. Slight bronchitis is often present during the first ten days, and may give rise to a troublesome cough, which is also frequently a result of pharyngitis. Though at first the patient generally has a heavy, inert appearance, in most cases of paratyphoid he looks and feels comparatively well by the second week, and he almost always feels still better at the end of the third week, even if the temperature is still raised. Only in a small proportion of cases of paratyphoid fever does the toxic condition with mental dulness, characteristic of severe typhoid fever, develop.

In 75 per cent of cases spots appear between the sixth and twelfth days. They come in crops, which last three or four days and are sometimes still visible after the temperature is normal. In paratyphoid they are often larger, more irregular in shape, more raised and of a deeper red colour than in typhoid fever, and they are sometimes remarkably profuse. They may not fade completely on pressure and sometimes leave a faint pigmented mark after they disappear. They occur most frequently over the lower ribs in front, on the flanks and on the back of the shoulders, but may even spread to the face, neck and arms, they are often arranged in groups. In very rare cases purpura occurs in the form of petechiæ over the abdomen and limbs, or of plaques the size of a shilling, especially on the arms or legs. The prognosis is then grave except in cases occurring during convalescence, when the purpura may be associated with swollen and bleeding gums and is doubtless the result of an insufficient diet.

The temperature rapidly rises at the onset, usually reaching its maximum in 48 hours. The morning and evening chart is of a typically spiky character with daily variations of at least two degrees, it is generally remittent, ranging between  $99^{\circ}$  and  $102^{\circ}$  during the second week, but it may be intermittent. Continued fever is generally present in the second week of typhoid fever but is very unusual in paratyphoid. In many cases of the latter the temperature does not reach  $103^{\circ}$ , and it is rarely higher than  $104^{\circ}$ .

The duration of fever varies between one and eight weeks in about half of the cases it is twenty days or less (Figs. 26 and 27). It generally terminates rapidly by lysis, which is often complete in

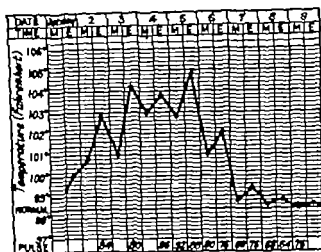


FIG. 26.—Short case of paratyphoid fever A.

two days. It may be followed by a sudden rise of temperature lasting for one to three days, and this is occasionally repeated once or twice. One, and occasionally two three, or even four true relapses, lasting six to fifteen days, with a return of pyrexia and

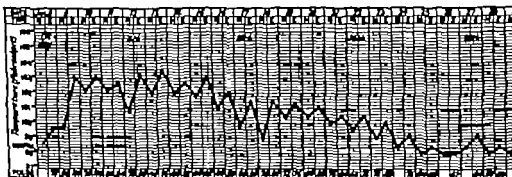


FIG. 27.—Paratyphoid fever B.

often with splenic enlargement and a new crop of spots, occur in about 10 per cent. of cases after between two and eighteen days, but most frequently after eight to ten days of apyrexia (Fig. 28). Rigors may be repeated throughout the illness in addition to the initial shivering attacks. Sweating is much more common in para

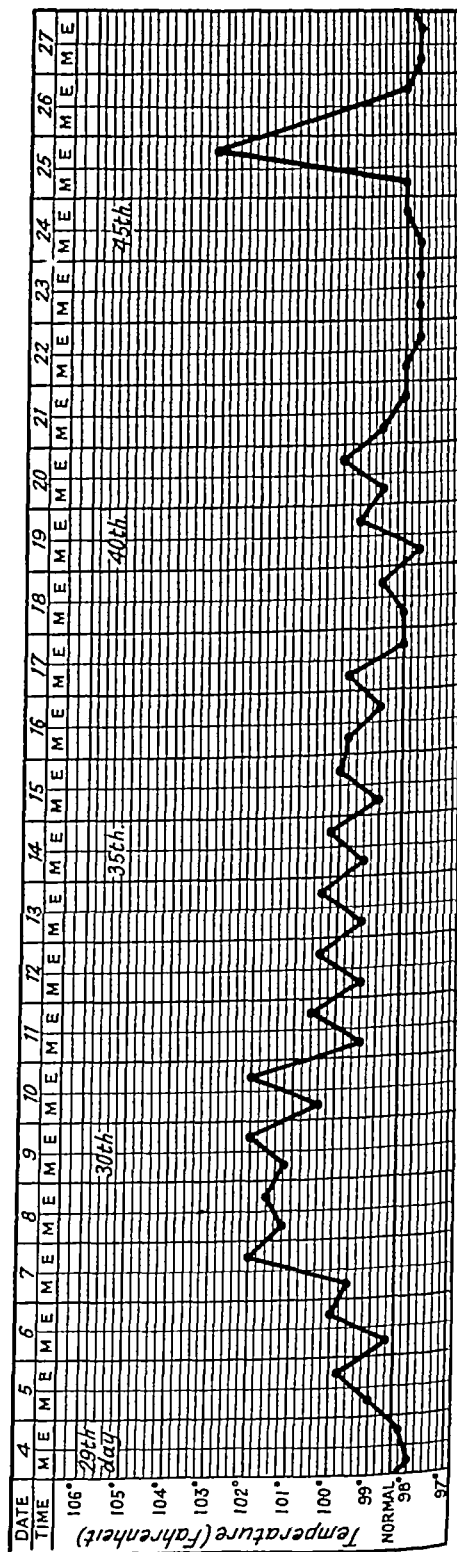
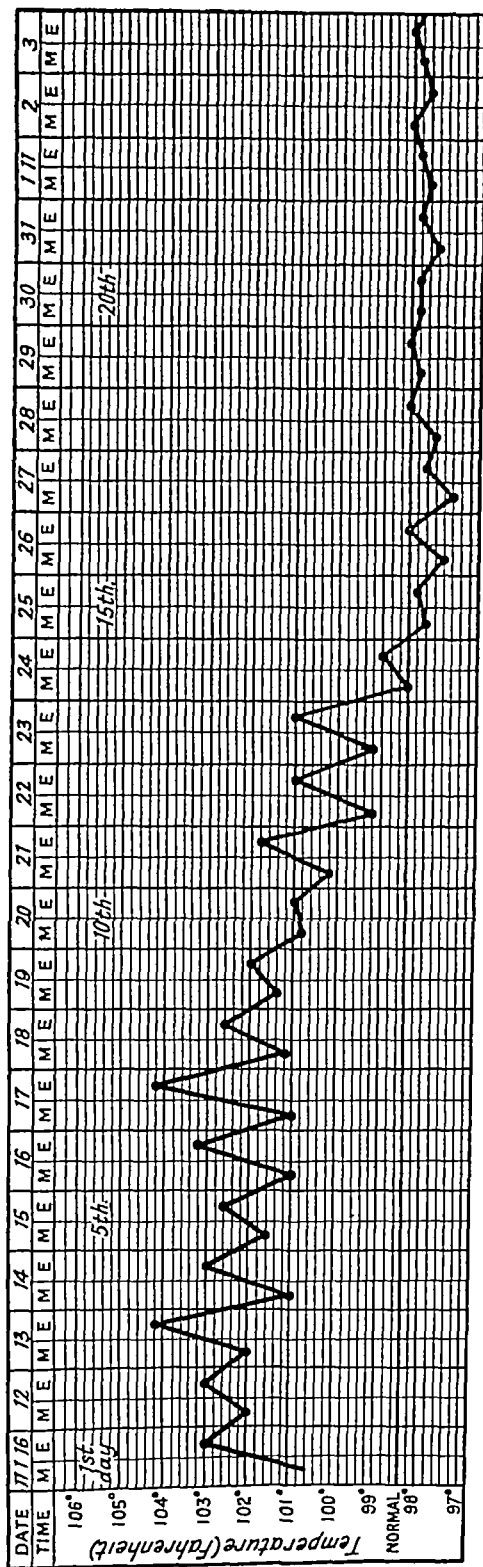


FIG 28 —Paratyphoid A with one long relapse and one short rise of temperature

typhoid than in typhoid fever but it generally does not begin until after the first few days of fever. It may be so profuse at night that the clothes have to be frequently changed, and sleep is consequently much disturbed.

A slow pulse is very characteristic of typhoid and paratyphoid fevers, a rate of 70 with a temperature of  $102^{\circ}$  being common at the onset however the pulse is often quite rapid. The rate sometimes varies considerably without obvious reason, and its variations are independent of variations in temperature. The pulse is very soft and often dicrotic, the systolic blood pressure varying between 80 and 105 mm. of mercury. Bradycardia may persist after convalescence, but it gives rise to no symptoms. In a number of cases the heart is slightly dilated with a systolic murmur at the apex slight cyanosis and coldness of the extremities are then observed, and tachycardia may be present and may persist for a considerable period, greatly delaying convalescence. If a patient with this condition returns to duty before the poisoned myocardium has completely recovered he is very apt to develop the effort syndrome (*vide* p. 203).

Hæmorrhage occurs in paratyphoid fever less frequently than in typhoid fever it is very rarely severe, but fatal cases have been recorded. Perforation, which had never been observed in paratyphoid fever before the war occurs in less than 1 per cent. of cases both of paratyphoid A and B generally in the fourth or fifth week the ileum, appendix and colon are involved with approximately equal frequency. In a third of the cases perforation is preceded by hæmorrhage. Meteorism is very rare and only occurs in the most severe and toxic cases.

Capillary bronchitis and broncho-pneumonia are comparatively common especially in bad weather. The latter is a serious complication and in my experience is more frequent in paratyphoid A than B it may develop at the onset, so that the case is at first regarded as simple pneumonia. In rare cases the broncho-pneumonia is complicated by a pulmonary abscess the *Bact. typhosus* and *paratyphosus* have been isolated from the sputum in pure culture. Miller (1917) observed a few cases in which paratyphoid fever appeared to activate latent pulmonary tuberculosis. Pleurisy, empyema, which is generally pneumococcal, infective endocarditis, and pericarditis have been observed in rare cases. Thrombosis may occur in the femoral and saphenous veins, especially of the

left side, or in the small subcutaneous branches of the internal saphenous vein over the calf Pharyngitis, tonsillitis and simple or ulcerative laryngitis may occur The latter may be due to perichondritis and lead to severe dyspnoea, which is relieved only by tracheotomy Laryngeal ulceration may produce stenosis, in which case all attempts to remove the tracheotomy tube are likely to fail. Transitory deafness is not uncommon and is probably due to mild otitis media. In several of the Salonica cases of paratyphoid fever pain was felt in the sole of the foot near the metatarsophalangeal joints, and there was tenderness in this region, though the joints themselves were not affected, the pain began towards the end of the illness and generally lasted about a fortnight. Miller observed several cases of arthritis, especially of the knee, two of which were followed by partial ankylosis. In several instances the joint had previously been injured The condition often relapsed, especially if the joint was strained In some cases true rheumatic fever complicated paratyphoid fever in men who had previously suffered from rheumatism

Slight albuminuria, which disappears when the temperature falls, is common; occasionally true nephritis with blood and casts in the urine is present, but cedema and other renal symptoms are rare. The blood disappears in a few days and the albumin does not remain much longer In a fatal case of paratyphoid fever I saw in Salonica I found the kidneys large and congested with numerous minute abscesses and hæmorrhages under the capsule, but the renal pelvis was normal Polyuria is common during convalescence, and in rare cases as much as ten litres of urine are passed in twenty-four hours for eight or ten days As typhoid and paratyphoid bacilli are often excreted in the urine, it is not surprising that pyelitis, cystitis, and in rare cases pyelonephritis may develop The infection generally begins about the third week, cystitis being in most cases secondary to pyelitis Orchitis is an occasional late complication The infection probably spreads from the urinary tract, which is always simultaneously infected The pus, which generally forms, always contains the specific bacterium The disease remains unilateral and no atrophy follows

The bile is always found to contain the organism after death, this accounts for the occasional development of cholecystitis Jaundice was a common complication of paratyphoid fever in Gallipoli, where epidemic jaundice was also very prevalent Its

rarity in France where it occurred in only 1·3 per cent. of enteric cases and was probably the result of subacute hepatic necrosis, and in Salonica, where epidemic jaundice was comparatively rare, makes it probable that the association was generally accidental. Suppurative pyelophlebitis is a very rare complication and is generally secondary to gangrenous inflammation of the appendix. Splenic abscess is a rare complication and results from infarction—it is almost always fatal, and the specific organism can be isolated from the pus.

Suppurative parotitis was a frequent complication in Mesopotamia in the hot weather when the vitality of the men was at a low ebb and the hospitals were understaffed. Suppurative myositis, thyroiditis, cerebral abscess, pericostitis and costal perichondritis, and suppurative peritonitis without perforation have been observed.

Intestinal symptoms may persist for many months after apparent recovery. Diarrhoea, worse after meals, the stools containing a little mucus, but no blood, generally alternates with constipation. Occasionally large quantities of mucus are passed, and a severer degree of constipation may be followed by an acute attack of pain with slight fever lasting two or three days, which may closely simulate appendicitis.

#### Bacteriological and Serological Diagnosis.\*

Although a diagnosis of "enteric fever" can often be made with almost complete certainty on clinical evidence alone, the only infallible proof that typhoid or paratyphoid fever A, B or C is present is the discovery in the blood, faeces or urine of the bacillus, with its characteristic cultural properties and its property of agglutinating with the specific immune serum obtained from an animal immunised with the organism in question.

From the bacteriological point of view the enteric fevers are nowadays taken as a single group. Following a report of a sub-committee of the International Society of Microbiology it has become customary to include under the title of the 'Salmonella Group' nearly fifty strains of bacilli known to cause either enteric fever or gastro-enteritis. Those responsible for the former are *Bact. typhorum* and *Bact. paratyphorum* A, B and C. True typhoid and paratyphoid B fevers are still endemic in Great Britain and paratyphoid A is endemic mainly in the East. Paratyphoid C infections may bacteriologically be divided into the West European

\* By F. A. Knott, M.D.

type, another name for the bacillus being the monophasic *Bact. supestifer*, and the Eastern or Hirschfeld type met with in eastern countries. Under war conditions any of these infections may spread beyond their usual geographical limits, although at present paratyphoid A and C are practically unknown in this country and in France. In this discussion of the bacteriological and serological diagnosis some account must therefore be taken of all these strains.

The direct methods of isolating the infecting organism are the same regardless of type. During the incubation period the bacilli pass *via* the intestinal lymphatics and mesenteric glands to the liver and spleen and are finally released into the blood stream, the initial bacteraemia coinciding with the onset of pyrexia. The clearing action of the reticulo-endothelial system is then brought into play, causing further concentration of the organisms in the liver and the now enlarged spleen. The bile becomes heavily infected and with it large numbers of bacilli are flooded into the intestine and make their appearance in the faeces. Meanwhile the intestinal lymphoid tissue has passed from the mildly to the acutely inflamed stage. While bacteraemia exists the bacilli are excreted in the urine, but after the blood stream has been cleared they generally appear only in the faeces, though occasionally they persist for many weeks in the urine of convalescent carriers. Thus in about 80 per cent of cases a positive blood culture can be obtained during the first five days of the illness, the frequency of positive urinary cultures is highest in the second week, and by the end of the third week the bacilli are plentiful in the faeces in most cases, but have disappeared from the blood stream and urine.

Specific agglutinins begin to appear in the serum during the second week of the illness. The titre rises for three or four weeks and then steadily falls during convalescence, some agglutinins remaining, however, for years.

In every case the help of the laboratory should be called in early. A blood culture should be made at once. Using the new cultural methods described below, positive results can often be obtained from the faeces within the first week. The popularity of the agglutination reactions is so great that in spite of the uncertainty introduced by the effects of T A B vaccine they are still often the only early tests made. It cannot be too strongly emphasised that blood and faecal cultures made without delay are under present conditions the laboratory methods of choice.

For blood cultures at least 10 c cm of blood should be withdrawn from a vein and transferred into eight to ten times its volume of culture media, which may be broth or peptone water preferably containing 0.5 per cent. of sodium taurocholate. Isolation of the bacilli from infected urine presents no particular difficulties, but contamination of the specimen should be carefully avoided. The percentage of positive cultures obtainable from the faeces rises from a very low figure in the first days of the illness to about 80 per cent. in the third week. By the seventh week it has fallen to 10 per cent. and about 2 per cent. of cases become carriers for a year or longer. At all stages excretion of the bacilli may be intermittent so that several faecal specimens may have to be examined. When the infective bacilli are present in very small numbers a drop of fresh faecal emulsion or a moist rectal swab may be planted first into liquid 'enrichment' broth containing one of various substances known to inhibit *Bact. coli*. Examples are 1 in 500 000 brilliant green, tetrathionate or brilliant green plus a small amount of Esbach's reagent. Overnight incubation considerably increases the proportionate number of enteric organisms present, and if a drop of this culture is plated serially upon MacConkey's bile-salt-lactose-agar litmus lactose agar or preferably Wilson and Blair's brilliant green-bismuth iron agar or the same authors desoxycholate-citrate-thiosulphate agar colonies of the pathogen are easily detected. The last-mentioned medium is based upon the observation by Laefson (1935) that sodium desoxycholate in the presence of sodium citrate inhibits the growth of *Bact. coli* leaving that of the intestinal pathogens largely unaffected. Both these Wilson and Blair media present difficulties in preparation, particularly under war conditions, but the Difco Laboratories of Detroit, U.S.A., have shown how this disadvantage can be overcome by supplying and exporting them in dehydrated form requiring only the addition of water before sterilisation and use. The Difco brilliant-green-bismuth-iron medium corresponds to Wilson and Blair's original formula the Difco SS Agar contains sodium desoxycholate and sodium citrate as well as brilliant green and sodium thiosulphate and corresponds to their second medium. There is no doubt that for mild infections convalescents and carriers the use of these enrichment methods is always desirable. After the final plating the fermentation reactions of any non lactose fermenting colonies are worked out and final identification of the strains may in the

case of the paratyphoid organisms require agglutination tests with specific anti-sera. These may be made (1) by the quick method of picking off a surface colony from one of the plates, mixing it with known serum on a microscope slide, and examining for agglutination with a low-power microscope; or (2) more accurately by Dreyer's method of making a series of serum dilutions, to each of which a little bacillary suspension is added in small glass tubes. These are partially immersed in a 57° C. water-bath and agglutination noted as flocculation visible to the naked eye.

In recent years study of the antigenic structure of the bacilli responsible for the enteric fevers has introduced complications into the performance of diagnostic agglutination tests. But in the process greater accuracy has been achieved. It is essential to consider not only the circumstances and period in which specific agglutinins in general can appear in the patient's serum, but also the significance of the detection of those concerned with particular antigenic fractions. All these enteric-producing bacilli may carry one or both of two antigenic complexes. In the flagella reside the H antigens and in the bacillary bodies the O or somatic antigens. The patient may develop agglutinins to one or both of these, and to investigate his reactions fully agglutination tests are now carried out with both H and O bacillary suspensions in all cases. Standardised suspensions can be obtained from the Oxford laboratories through the Medical Research Council; they are so graded that the final results are quantitatively comparable, a great advantage over the earlier methods.

Interpretation of diagnostic agglutination tests depends upon whether the patient has previously suffered from enteric fever or received prophylactic inoculations of T A B vaccine. In the absence of either of these conditions there is a tendency for a patient infected with typhoid fever to develop stronger typhoid O than H agglutinins, whereas with paratyphoid infections the H titres tend to be higher than the O. Numerous exceptions are met with, however, and it is unsafe to base a bacteriological diagnosis upon this difference alone. In practice it is best to observe the rate at which both types of agglutinins appear in the serum. Discovery that a patient has developed strong O agglutinins to any of these bacilli is of great value in indicating a true enteric infection. The exact type of organism causing infection may not be indicated by the O estimation, because cross agglutination is so marked with O sus-

penansons of the different strains. But with the H suspensions, which are much more specific although the H agglutinins are often slower to develop differentiation can usually be made. A modern agglutination test therefore consists of mixing the patient's serum in graded dilutions with a series of known H and O bacillary suspensions, prepared from strains likely to be met with in the district. Whenever possible the test is repeated at intervals. Early positive O agglutination affecting several strains suggests an enteric fever, and a rising H titre towards one particular strain incriminates it as the infecting agent.

The interpretation of agglutination reactions may be complicated by two circumstances. The patient may come from a community having some natural immunity and therefore some natural agglutinins in his serum and he may have previously received prophylactic inoculations such as T.A.B. vaccine. Where enteric fevers are endemic, subclinical infections often result in high agglutinin titres. But following infection the original O titre falls much more rapidly than the H, and therefore the residual agglutinins are mainly of the H type. In such a district a high O titre is particularly to be sought before a diagnosis of new enteric infection is made. Prophylactic T.A.B. vaccine injections at first provoke both H and O agglutinins, but in this case also the O tend to disappear rapidly and therefore, although the patient may for years carry agglutinins in his serum for all the types of bacilli included in his original vaccine these will all be of the H type. Consequently here again for proving recent infection most diagnostic significance attaches to the reappearance of O agglutinins and particularly to a steady rise in their concentration.

A further difficulty may arise. In a patient, who owing to previous subclinical infection or inoculation carries these agglutinins in his serum, some other infection may cause a temporary non-specific rise—the anamnestic reaction. But the rise is rarely very marked and is never as specific or as continuous as in enteric fever. Moreover in this case also it is normal for only the H agglutinins to be affected—another instance of the great diagnostic reliability of observations of the O titre.

Regarding the question as to what is a clinically significant serum dilution at which positive agglutination can be obtained, great difficulties were encountered in the past because the case with which different bacillary suspensions could be agglutinated varied con

siderably. With the modern standardised suspensions a factor is given by which the actual limiting dilution must be reduced to give a corrected reading. If this corrected limiting detection is 1 in 100 or higher it is considered to be significant, but it is to be remembered that continuation of such a rise is of even more clinical importance. The Dreyer 57° C water-bath technique of performing these standardised agglutination tests has served well for many years and will continue to do so.

Recently two further bacteriological tests have been devised for enteric fever once the infecting organism has been isolated in culture. The first concerns the discovery by Felix that virulent strains of typhoid bacilli carry, apart from elements such as the H and O already noted, a particular antigenic component now known as the "virulence or V<sub>1</sub> antigen". Formolised bacillary suspensions containing only V<sub>1</sub> and devoid of H and O have been prepared, so that a patient's serum can now be tested by agglutination reactions for the presence of V<sub>1</sub> agglutinins, i.e. for the result of contact with *virulent* typhoid bacilli. The method seems likely to be valuable in detecting recent carriers, but limitations are imposed by the fact that the V<sub>1</sub> agglutinins disappear from the serum relatively quickly. A therapeutic antiserum for use in typhoid fever based upon products of a high titre for both O and V<sub>1</sub> antigens is available.

Epidemiological investigation of typhoid outbreaks is now unquestionably improved by the isolation by Craigie and Yen (1938) and Felix (1943) of bacteriophage, which acts specifically upon different virulent strains of typhoid and paratyphoid B bacilli (Felix and Colln, 1943). At present at least thirteen different phage-types of *Bact. typhosum* have been identified. Once a set of these phages is made, it is necessary only to prepare a series of simple surface growths of the bacillus to be tested, a drop of each phage being allowed to dry on an area of growth. Two hours' incubation overnight, storage in the cold and short further incubation at 37° C will produce a patch of lysis in the sample of culture which is in contact with its appropriate phage. The phage type of any strain is found to be extremely constant, so that in this test there is a means of connecting a carrier with particular outbreaks or distant cases, following the separate courses of coincident mixed epidemics, and in fact tracing any lines of typhoid spread whether among patients or food products. Bradley (1943) and Hutchinson (1943)

have reported the successful use of phage typing in the investigation of sporadic cases of typhoid and paratyphoid B fever

Leucopenia is common but is less constant in paratyphoid than in typhoid fever especially in the early stages and in mild cases thus between 8,000 and 14 000 leucocytes were present per c.mm. in several cases examined by Elworthy in Salonica. Complications such as broncho-pneumonia give rise to definite leucocytosis. A relative increase in lymphocytes is present in a larger proportion of cases Elworthy rarely found less than 36 per cent. even when the total count was as high as 14 000 Such a differential count is not of course specific, as it is quite compatible with various other infections.

### Differential Diagnosis

Paratyphoid fever differs chiefly from typhoid fever in being a much milder infection with a much smaller mortality but these differences may be less marked in patients who have received anti typhoid inoculation. The profound toxic state, which is comparatively common in unmodified typhoid fever is only seen in exceptional cases. Whereas in typhoid fever the patient tends to become more and more toxic as the disease progresses, in paratyphoid fever he is generally at his worst at the end of the first week, and even in severe cases, which give rise to the greatest anxiety in the early stages, remarkable improvement occurs before the temperature falls, and convalescence is unexpectedly rapid. The onset is more acute, and the temperature rises more rapidly remains raised for a shorter period and shows greater daily variations in paratyphoid fever and there is rarely any period of continued high fever, such as is seen in the second week of typhoid fever Shivering at the onset is much more common in paratyphoid than in typhoid fever and rigors in the course of the illness are not so rare. The spleen is firmer and is generally more tender and the spots are often larger more irregular in shape and more raised.

Many cases are at first diagnosed as *influenza*, especially when the onset is acute, and for a few days it is often quite impossible to distinguish between the two diseases. True *influenza* was, however comparatively rare in the last war until the pandemic at the end of 1918 and catarrh of the upper respiratory passages is almost always present. Apart from the bacteriological evidence leucopenia with relative lymphocytosis points to enteric fever though its absence does not exclude the diagnosis the spleen is always either

palpable or is found to be enlarged on percussion, but this is sometimes also the case in influenza. The temperature generally falls before the end of the first week in influenza, and the diagnosis is often settled about this time by the appearance of spots.

The onset of both forms of *trench fever* may simulate that of enteric fever, but the course of the illness and the pain and tenderness of the shins quickly indicate the nature of the infection. The temperature chart in prolonged cases of typhoid and paratyphoid fever may resemble that of *Malta* or *abortus fever*, in doubtful cases the diagnosis can be settled only by blood culture and agglutination reactions.

Exceptional cases of enteric fever, in which the onset is very acute with diarrhoea, vomiting and abdominal pain, may closely simulate *food poisoning*, the course of the disease, the presence of spots, the enlargement of the spleen, and the bacteriological examination will settle the diagnosis. When abdominal pain and vomiting occur at the onset, especially if there is tenderness in the right iliac fossa, *appendicitis* may be simulated, but instead of the leucopenia with a relative increase of mononuclear cells seen in typhoid and paratyphoid fever, leucocytosis with a relative increase of polymorphonuclear cells is present in appendicitis, and in most of these paratyphoid cases the suspicious symptoms rapidly disappear, though the temperature remains raised. As the appendix is often involved in the inflammation and ulceration of the bowel, it may give rise to peritonitis both with and without perforation. In two out of Miller's 500 cases of paratyphoid fever an acutely inflamed appendix had to be removed, in both cases the infection was due to *Bact paratyphosum B*. The early joint pains are in rare cases so severe that *rheumatic fever* is suspected, the joints are, however, never swollen, and the temperature does not at once fall to normal with salicylates. In exceptional cases *dysentery*, *cholera* or *meningitis* may be so closely simulated that the possibility of enteric fever is not considered until the bacteriological examination of the stools and cerebro-spinal fluid demonstrates the absence of the specific organisms of these diseases and the presence of the *Bact typhosus* or *paratyphosus*. The possibility of a double infection with dysentery and enteric fever must also be remembered in war time, and this association was not uncommon in Gallipoli.

When the initial fever is accompanied by shivering followed by profuse sweating *malaria* is simulated, but the temperature

does not fall with the sweating. The sub-tertian form of malaria may resemble enteric fever very closely. In such cases the differentiation can be made only by a blood examination, as remissions may be slight, and typical rigors, whilst not uncommon in typhoid and paratyphoid fever do not always occur in malaria. The intense headache and high fever in enteric fever beginning acutely often led to a diagnosis of *heat stroke* in Mesopotamia.

It is impossible to distinguish clinically between paratyphoid A, B and C, and the majority of the features which have at different times been said to be characteristic of one or other condition have not proved to be so on further investigation. This is particularly noticeable when the descriptions of the disease as seen in different areas during the last war are compared. Thus I found that none of the symptoms, which were said by observers in France to aid in the diagnosis, were of any value in Lemnos or Salonica. It has been repeatedly stated that paratyphoid A is a much milder disease than paratyphoid B but this was not my experience as many of the most severe cases as well as some of the least severe were examples of paratyphoid A. The only symptom which seemed to me to be generally distinctive was the very large and very hard spleen sometimes seen in paratyphoid A.

### Prognosis

The prognosis in paratyphoid fever is very much better than that in typhoid fever unmodified by previous inoculation. The mortality among 2 118 bacteriologically proved cases in France up to August 25th, 1916 was only 29 or 1.32 per cent. In the same period 166 out of 1 501 cases of typhoid fever died of these 903 were inoculated and 47 died (5.2 per cent.) and 508 had not been inoculated and 119 died (23.4 per cent.) The mortality was probably rather higher in the Mediterranean Expeditionary Force, though not in Salonica. In Mesopotamia it was over 10 per cent. but this was largely due to the great heat in July 1916 when 30 out of 39 deaths, mostly due to hyperpyrexia and cardiac failure, occurred in the cases seen by Batt and Feiling. When at the end of the war universal inoculation against paratyphoid as well as typhoid fever was practised the severity of the disease greatly diminished and the mortality fell still lower.

The chief causes of death are perforation broncho-pneumonia, profuse hæmorrhage, and toxæmia, which is also an important factor in many cases of death occurring from other causes. So far

as my experience goes, and it agrees with the *post mortem* records of Dawson and Whittington, the extreme end of the ileum was most severely ulcerated in cases in which toxæmia was the main or sole cause of death. The ileo-cæcal aperture was so much involved that there appeared to be some obstruction due to swelling of the mucous membrane, and to this might be added a spasm or absence of the normal periodic relaxation of the ileo-cæcal sphincter similar to that which occurs in acute appendicitis. This would result in severe ileal stasis and consequent intestinal intoxication, which would aggravate the toxæmia due to the specific infection. Death has also resulted from abscess of the spleen, gangrenous cholecystitis and peritonitis without perforation. I performed the autopsy on a case of paratyphoid A at Salonica, in which death occurred from suppurative nephritis in the fifth week, when all the ulcers in the intestine had healed; the organism was isolated from the bile obtained from the gall-bladder.

The proportion of severely toxic cases in paratyphoid fever is small, and ambulatory cases lasting only a few days with mild pyrexia, slight headache, splenic enlargement and perhaps spots occur, though their frequency is not known, as many doubtless escape recognition. Even in the worst cases convalescence is comparatively rapid if an adequate diet is given. The slow convalescence with prolonged gastro-intestinal disturbance, furred tongue, anorexia, epigastric fulness after meals, liability to vomit, and tenderness over the liver and gall-bladder, which have been described as almost constant by French authors, are undoubtedly due to the milk diet they invariably prescribe.

The faster the pulse in relation to the temperature the more severe is the infection, a rate of over 100 after the fifth day indicating a case of some severity. Severe bronchitis, much mental clouding, and abdominal distension are the most serious symptoms. The prognosis is much better in warm than in cold weather, when the disease is very liable to be complicated by broncho-pneumonia and relapses are more common, and in very hot weather, when the patient is likely to be in a debilitated condition.

### Prophylaxis.

The prophylaxis of typhoid and paratyphoid fever consists in the protective inoculation of all soldiers and the prevention of contamination of the water supply and of food with infective material. The measures required are the same as those described

on p 309 for the prophylaxis of amoebic dysentery Every suspicious case should be sent into hospital without delay Familiarity with the clinical features of the disease will lead those in charge to call in the aid of a bacteriologist at an early stage, when diagnosis by blood culture is possible. As soon as the diagnosis is made, the man's unit should be informed, so that his bedding may be disinfected and a search made for the source of infection. There is no real need to isolate cases in special hospital wards, though it may be convenient to do so especially if inexperienced orderlies are in charge. The faeces and urine should be disinfected immediately and incinerated as quickly as possible. As the sputum may also be infective, it should be disinfected and patients should not be allowed to cough into the air but into paper handkerchiefs which can be burnt It is advisable to isolate convalescent patients until their stools and urine are proved to contain no typhoid or paratyphoid bacilli on three consecutive occasions at intervals of a week, in order to avoid sending carriers back to their units. If it were not for this, a man who has had paratyphoid fever would often be fit for light duty a month after his temperature has fallen to normal. Mild cases which have escaped recognition, are a far greater danger as a source of infection than convalescent carriers. Their number is likely to increase considerably now that the disease is so much less severe as a result of mixed typhoid and paratyphoid inoculation.

Inoculation against typhoid fever was first attempted by Sir Almroth Wright in 1896 and was used on a small scale in the South African War with promising but inconclusive results. In the following years it proved extremely effective in the Indian Army, and there is no doubt that the almost universal inoculation practised in the last war is the explanation of the very small number of cases which occurred. The official returns in the war of 1914-1918 showed that the admission rate for typhoid fever amongst the troops in France who had not been inoculated was fifteen times as great as amongst those who had been inoculated, and the death rate was seventy times as high.

If a mixed typhoid and paratyphoid vaccine had been used from the beginning of the war, there would probably have been only 75 cases of paratyphoid fever instead of 2,118 among the British troops in France during the first two years of the war or about 5 per cent. of the number of cases of typhoid fever as this appears

to be the relative frequency of the fevers in the uninoculated civil population. After January 1916 the triple vaccine, known as T.A.B., became the only official vaccine for the British and French Armies and proved most effective in preventing paratyphoid as well as typhoid fever. The vaccine now in use consists of 500 millions *Bact typhosus*, and 250 millions each of *Bact paratyphosus A* and *B*, prepared from virulent strains, and two doses are given with an interval of a week. This vaccine gives some protection against paratyphoid C owing to the antigenic similarity of the latter to A and B.

It is interesting to note how universal inoculation, an improved vaccine, and better sanitation continue to reduce the incidence of the enteric fevers. In the four years 1928 to 1932 there were 1,057 cases of typhoid, 302 paratyphoid A and 53 paratyphoid B in the British Army in India compared with 354, 223 and 9 respectively in the following five years. But it would be unsafe to assume that systematic use of T.A.B. vaccine will eliminate any of these fevers from the Army, especially under war conditions, because the protection is only relative, and the civilians amongst whom the soldiers live are not inoculated. Moreover, T.A.B. inoculation is still not compulsory in the British Army, and consequently between 5 and 10 per cent of soldiers are not immunised. In spite of this no case of enteric occurred in the B.E.F. in France in 1939 and very few in 1940. The total number of cases in the B.E.F. and Home Forces in the first twelve months of war was 32, of which 26 had not been inoculated. From these figures it can be calculated that the chances of contracting typhoid or paratyphoid fever is increased about seventeen times by refusing inoculation. Typhoid fever and paratyphoid A were very common among Italian prisoners in the Middle East, though very rare among British prisoners in Italian hands, in spite of living in a camp in which sanitation was of a primitive type and where typhoid was rife among their captors. This proved to be due to the potent vaccine used for British troops in contrast with the Castellani and other Italian vaccines which appear to have been prepared from non-virulent strains (Boyd, 1943).

Universal immunisation is unnecessary for the civil population in war conditions, as immunisation during an epidemic gives the same degree of protection as in non-epidemic periods, so that inoculation can be reserved for groups of people who are at any time endangered

### Treatment

Ever since a visit to America in 1906 when I saw the remarkably good effect of a generous diet in shortening convalescence and in reducing the liability to septic complications without increasing the danger of hæmorrhage or perforation, I have treated all enteric patients in this way. I have never had occasion to regret it, nor have I ever heard of any ill-result. It is the duty of those in charge of sick soldiers to make them fit for active service as quickly as possible, and I am quite certain that a diet consisting at least of milk-puddings, custard, coddled eggs, bread, toast and biscuits with butter and honey or fruit jelly, and strained orange juice in addition to milk makes a man fit for duty very much sooner than he would be on a semi-starvation diet of milk alone. Such articles of diet are completely fluid by the time the ulcerated area of the ileum and colon is reached. On the other hand, I have several times seen curds in these parts of the bowel after the death of patients who had been on a purely milk diet. Biscuits, which the patient has to chew are of the greatest value, as they stimulate the secretion of saliva and keep the tongue moist. I have never seen the so-called typhoid tongue in a patient treated in this way. It is simply a result of a diet which requires no chewing and which calls forth no secretion of saliva. Complications such as parotitis are directly due to a septic condition of the mouth, which can easily be prevented by good nursing and an antiseptic alkaline mouth-wash if a suitable diet is given. Galambos (1917) treated about 2 000 Austrian patients with a liberal diet containing 2 500 to over 3 000 calories and found that such complications as empyema, parotitis, otitis, thrombosis, neuritis and abscess formation, which were comparatively common early in the war became extremely rare. There were, for example, only two cases of parotitis out of 2,000 patients, and in no case could hæmorrhage or perforation be attributed to the diet. The percentage of relapses fell from 10 to 3 or 4. An abundant supply of fluid should be given, and in severe toxic cases normal saline solution should be injected intravenously by the drip method. Tepid sponging, repeated every four hours, is very useful when the temperature exceeds 103° and when the patient is restless or delirious.

The only drugs which require consideration are hexamine and the sulphonamides. Hexamine is a powerful urinary antiseptic and is a biliary antiseptic as well. As the chief source of the organism in

carriers is the biliary tract, the number of convalescent carriers would be very greatly reduced if it were possible to keep it sterile by the systematic use of hexamine. At the same time it diminishes the liability to cholecystitis and cystitis. The drug should be given in doses of gr 15 three times a day until the patient leaves hospital. This dose is much smaller than that used in the treatment of cholecystitis, but doses larger than 15 or 20 grains give rise to cystitis unless the urine is kept alkaline by the addition of sodium bicarbonate and sodium citrate in order to prevent the liberation of formalin in the urine, unfortunately the antiseptic action in the urinary tract is thereby prevented. But a comparatively small dose of hexamine may be sufficient to prevent infection of the biliary tract, though it cannot overcome an infection when it has once been established. If acute cholecystitis develops or the patient becomes a carrier, 100 grains of hexamine with 80 grains each of sodium bicarbonate and sodium citrate in 2 ounces of water should be given after breakfast, after tea and at 10 p.m. after a glass of milk for six weeks in order to sterilise the biliary tract.

Typhoid and paratyphoid fevers are uninfluenced by sulphaguanidine or any other of the sulphonamide group of drugs, but sulphapyridine acts almost as a specific in carriers with persistent infection of the urinary tract. This is due to the fact that the *Bact. typhosus* is only slightly affected by sulphonamides in therapeutic concentrations in the blood, but is killed by the concentration attained in the urine, which often amounts to ten or twenty times that in the blood (Thrower, 1941).

Vaccines are useless for treatment, but in typhoid fever Felix's vi-serum given early and repeatedly in full doses frequently influences the toxæmia.

The patient should not be allowed to get up, however slight the illness may have been, until his temperature has been normal for a week. I have seen fatal perforation occur in a case of paratyphoid. A just when defervescence appeared to be almost complete, and at the autopsy numerous ulcers, only a few of which showed signs of healing, were still present, so that a fall in temperature does not always indicate that the ulcers have healed.

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## CHAPTER XXI

### DYSENTERY

#### BACILLARY DYSENTERY

Bacillary dysentery has always been common in armies on active service. Out of 30,000 British soldiers who fought in the Crimea 7,883 suffered from dysentery and of these 2,143 died. In the South African War there were 38,108 cases with 1,342 deaths.

In the last war bacillary dysentery made its first appearance in 1914 in East Prussia and Galicia and among von Kluck's ill-fed and tired soldiers on their march towards Paris. It headed the list of infectious diseases in the German army with 155,000 cases. It did not attack British troops in France and Flanders until July 1916. It disappeared in the winter and recurred in the autumn of 1917 and 1918. Its maximal incidence in the British Army in France was 126 cases per 100,000 in September 1916, and throughout it appeared in a mild form. On the other hand, it proved a very serious source of invaliding in Gallipoli, Mesopotamia, Salonica and Palestine, there were no less than 2,900 cases per 100,000 men in Mesopotamia in 1916. In the East Shiga and Flexner infections were about equally common, but 85 per cent of cases in the West were of the Flexner type. The mortality was low in every theatre of the war, the maximum being at Gallipoli, and even there it was only 5 per cent.

As bacillary dysentery has been endemic in France and in Great Britain since the fourteenth century at least and mild and scattered epidemics still constantly occur in warm weather, it seemed likely that it would attack British troops in France again in the present war. But the hygiene of the Army was so good that only seven cases of probable dysentery of non-specific types had been observed in the B.E.F. in France by January 20th, 1940, and very few additional cases occurred before the evacuation.

A severe epidemic of bacillary dysentery, caused by a virulent Shiga infection, occurred among exhausted German soldiers in the autumn of 1939 in Poland, where the disease is endemic. The disease quickly spread to Germany (Gantenberg, 1939, Kalk, 1940). There were no cases of amoebic dysentery.

The incidence of dysentery has been remarkably low among

British troops in the Middle East. This has been largely a result of educating them by means of propaganda and of a vigorous anti fly campaign. About 40 per cent. of cases were caused by infection with Flexner's bacillus, 30 per cent. with Shiga's and 5 per cent. with Sonne's, the remainder being amoebic, but many cases of bacillary dysentery which did not get well quickly or relapsed were found to be infected with amoebae as well as *Bact. dysenteriae*.

### *Ætiology*

House-flies are the chief means of spreading both forms of dysentery. How they do this is described in detail in the chapter on amoebic dysentery (p. 303).

Owing to the extreme frequency of defecation soiling of the patient's clothes and skin is common and hospital attendants may infect themselves if they are careless about washing their hands. Defective personal hygiene is a frequent cause of the spread of the disease in camps, as the hands are likely to be soiled with infective material in the latrine, and if they are not thoroughly washed food becomes contaminated.

The *Bact. dysenteriae* is rapidly destroyed by dilute hydrochloric acid. A normal secretion of gastric juice is therefore likely to protect against infection. Camps (1933) found that 14 per cent. of men who had suffered from bacillary or amoebic dysentery had achlorhydria and 8 per cent. had hypochlorhydria compared with 4 and 1 per cent. respectively of normal men of the same age and the combined observations of Miano and Forza showed that 59 per cent. of 37 patients actually suffering from amoebic dysentery had hypo- and achlorhydria (Forza, 1939). The achlorhydria and hypochlorhydria were probably a predisposing cause and not a result of the dysentery. I have known patients, who had lived for many years in the tropics without developing dysentery contract the disease after achlorhydria had developed, in one case as a result of alcoholic excess, in another after treatment with arsenic for post-malarial anaemia, and in a third after a gastrojejunostomy performed for a duodenal ulcer. These observations strengthen the suggestion I made in 1915 that one factor in the high incidence of dysentery among the troops at Gallipoli may have been the prevalence of a temporary achlorhydria resulting from privation and poor food.

Infection may at once lead to an attack of diarrhoea or dysentery or the individual may become a carrier and either remain healthy

though still a carrier, or he may eventually develop dysentery if, as a result of slight food poisoning or some intercurrent mild infection, the resistance of his intestinal mucous membrane is temporarily lowered. The stools of convalescent patients may still contain the bacilli even a year after infection. So-called "contact carriers" have generally had mild diarrhoea, the nature of which has not been recognised, as there is no doubt that infection with a dysentery bacillus may lead to nothing more severe than a slight catarrhal colitis, in which loose stools containing no blood and little or no mucus are passed. This is particularly true in the case of infection with the Sonne bacillus, which rarely gives rise to the typical "bloody flux" of classical dysentery.

### **Morbid Anatomy.**

In acute bacillary dysentery all or nearly all of the colon and the last one to three feet of the ileum are involved. In chronic cases the changes are generally confined to the lower half of the colon.

The disease begins as an acute inflammation of the whole mucous membrane with necrosis involving the lymphoid follicles, which later give rise to superficial ulcers, the submucous tissue and the muscular and peritoneal coats escaping more or less completely. In a typical specimen dark greenish grey necrotic areas and irregular superficial ulcers are scattered over the inflamed and thickened mucous membrane. Perforation and peritonitis hardly ever occur. When the disease becomes chronic, the necrotic areas are completely replaced by ulcers, which may be so extensive that only scattered islets of thickened mucous membrane remain. Felson (1941) has described the development of the lesion as seen from day to day with the sigmoidoscope. On the first day the mucous membrane is already acutely inflamed—red, glistening and cedematous, covered with mucus, and "weeping blood". Punctate follicular hyperplasia is already present, the enlarged solitary lymph nodes standing out prominently like grains of sand on a red background. On the second or third day the centre of the follicles undergoes necrosis to form minute ulcers, after which the discrete areas of necrosis and ulceration spread and become confluent. The appearance is totally unlike that of amoebic dysentery, in which the mucous membrane surrounding the actual lesions is normal in appearance.

### **Symptoms.**

The incubation period is between one and seven days. The

disease generally begins acutely with griping abdominal pain, this is soon followed by the passage of fluid stools containing mucus which may be blood-stained from the onset. In the early stages between six and thirty stools, many of which consist entirely of mucus mixed with pus and blood, are passed in the twenty four hours. When the rectum is involved tenesmus occurs in addition to the abdominal pain which generally precedes defecation, it may be so persistent that the patient has a constant desire to open his bowels and is unwilling to leave the bed pan. In severe cases there is often incontinence of faeces. The anus may become inflamed, excoriated and painful. Drinking, eating and every movement intensify the desire to defaecate. Micturition may also be frequent and painful. The patient rapidly becomes exhausted, and a bluish-red flush often appears on the cheeks. Moderate fever is generally present, and the temperature may rise as high as  $105^{\circ}$ . The pulse is rapid and weak.

As a rule nothing abnormal is found on abdominal examination. There is generally no tenderness, but occasionally the iliac colon forms a contracted, freely movable and slightly tender cord. In very severe cases there may be widespread tenderness and rigidity.

In mild cases the acute symptoms do not last more than two or three days. In severer cases improvement begins only after a week or fortnight, and convalescence is slow owing to the great weakness which results from the diarrhoea and toxæmia. The disease sometimes becomes chronic diarrhoea persists with the passage of blood and mucus or of abundant thick mucus with little or no blood, and emaciation, weakness and anaemia become progressively greater. In most cases of this kind the patient ultimately recovers but death may result from exhaustion. An irritable state of the bowels, which may last for many years often follows recovery from the acute infection (*vide p 320*).

An individual, who has previously been subject to hæmorrhoids, is very liable to be troubled with bleeding and prolapse after an attack of dysentery. The possibility of this source of hæmorrhage, when the stools have become solid, should be borne in mind.

Toxic nephritis has been a frequent complication of bacillary dysentery in the Middle East during the present war (Boyd and Fairley 1942).

Arthritis involving one or more of the large joints may occur. It is common in some epidemics, such as that which attacked the

British troops in the South African War, but was comparatively rare in the last war, except in a localised epidemic in the French Army during 1916. It was a common complication in the epidemic among German soldiers in Poland in 1939 (Gantenberg). The symptoms generally do not develop until the diarrhoea has ceased for an interval varying between a few days and three weeks. Their severity does not vary with that of the original attack, and arthritis is not uncommon after an attack which was so slight that its nature was not recognised at the time, and the patient may not even have gone into hospital for it. The possibility of a dysenteric origin should therefore be considered in every case of arthritis when bacillary dysentery is prevalent. The knee is most often affected, but several joints may be involved simultaneously. There is a large effusion with considerable muscular atrophy, but less pain, tenderness and redness than in acute rheumatism, the disease bearing more resemblance to gonococcal arthritis. Suppuration never occurs. Complete recovery generally takes place, but sometimes not until several months have elapsed. In rare cases permanent crippling, which resembles that following rheumatoid arthritis, may follow. Fever is common, but the heart is never affected. The effusion into the joint is sterile, but contains numerous polymorphonuclear cells. I have seen two cases in which an attack of dysentery brought on acute gout in men who were already subject to the disease. It is necessary to distinguish dysenteric arthritis from the acute synovitis which may follow the injection of anti-dysenteric serum. The latter is often accompanied by other symptoms of serum disease, such as urticaria and headache, and generally begins between the fifth and tenth day after injection.

### Diagnosis.

The only conclusive evidence of the presence of bacillary dysentery is the discovery in the faeces of the bacillus with its characteristic cultural properties and its agglutination with specific immune sera. A piece of mucus free from faeces should be picked out of the stool and sent at once for immediate examination, as the organism can be much more easily isolated from such a specimen than from the actual faeces. According to Friedmann the percentage of positive findings was increased from 20 to 100 per cent. by examining the mucus obtained directly from the pelvic colon through a sigmoidoscope. Similarly Manson Bahr found that whereas the first plates made from stools kept in enamelled bed-

pans with well-fitting lids and kept in a warm place gave almost pure cultures of *Bact. dysenteriae*; not a single colony could be recovered from plates sown from the same stools at the end of eight hours. A definite result cannot be obtained in less than three days but a presumptive diagnosis can often be made in thirty-six hours.

The macroscopic and still more the microscopic appearance of the stools of bacillary dysentery are so characteristic that a probable diagnosis can be made without waiting for the bacteriological report (Wenyon and O'Connor 1917). Glairy white or yellowish mucus streaked with bright red blood is passed alone or with a little faecal matter between the patches of mucus. Under the microscope red corpuscles are seen scattered over a ground of polymorpho-nuclear pus-cells, mixed with a few larger round mononuclear cells and a still smaller number of very large cells, which have occasionally been mistaken by inexperienced observers for amoebae. All these cells are phagocytic, and they can be found in the submucous tissue of the colon after death. The characteristic stools of the acute attack are followed by the passage of brown liquid faeces which contain similar cells to those found in the mucus. When the stools become formed the faeces are at first coated with mucus, which contains numerous elongated cells derived from the columnar epithelium of the mucous membrane a few of which are sometimes present during the acute stage. In some cases small pieces of necrotic mucous membrane are passed.

The stools in amoebic dysentery are generally less copious and watery than in bacillary dysentery. The blood and mucus are generally darker the blood being often brown or even black and the mucus transparent and dark brown. The mucus is more often mixed with faeces, and in some cases the stools consist simply of unformed faeces impregnated with mucus. In addition to the amoebae there are often numerous red corpuscles, but comparatively few intact pus-cells. Most leucocytes and tissue cells show serum cytolytic changes and are represented by single nuclei with little or no cytoplasm.

Bacillary dysentery differs from amoebic dysentery in the more acute onset, higher fever and severer toxic symptoms. Tenesmus is more frequent and thickening of the colon is much less common. Tenderness is less marked, and when present is generally greater on the left side than the right. Hepatitis and hepatic abscesses never

occur, and local and general peritonitis are very rare. There is no leucocytosis, a greater number of leucocytes than 15,000 per cubic mm pointing strongly to amoebic dysentery.

In the epidemic in the Mediterranean Forces in 1915 the clinical picture was much confused by the frequent association of amoebic with bacillary dysentery. The possibility of such an association should be considered whenever a case of apparently amoebic or bacillary dysentery does not respond to treatment with emetine or anti-dysenteric serum respectively. In other cases dysentery was associated with paratyphoid fever and with infective jaundice.

In rare cases the onset is so acute and the stools are so watery and profuse that the disease may be mistaken for cholera, until large quantities of mucus with or without blood are also passed and the diagnosis becomes clear. Most cases of so-called malarial dysentery are really due to mixed infection. In all but 2 of 68 cases investigated at Salonica, in which malaria was associated with hæmorrhagic diarrhoea, Graham (1918) found dysentery organisms. In spite of this most cases of "malarial dysentery" respond promptly to anti-malarial treatment.

### **Bacteriological and Serological Diagnosis.\***

Early studies of bacillary dysentery showed that the organisms responsible fell into two main groups. Bacilli of the Flexner group caused the majority of cases in Western Europe, and cases due to the more toxic Shiga strains predominated in the East and sub-tropical zones. There are sharp cultural and serological differences between the typical Flexner and Shiga groups, and their identification in the laboratory presents no special difficulty. But in recent years epidemics of dysentery have been shown to arise from bacilli, which fail in a few biological properties to correspond exactly to either of the classical types. The disease caused by these atypical organisms is rarely as severe and the epidemics are not as widespread as in the case of true Flexner or Shiga dysentery, but the pathology is the same in all and the same principles are to be adopted in treatment and control.

It is characteristic of the two classical strains that the bacilli having no flagella are non-motile, that they produce no gas when fermenting sugars, form little or no indole when growing in broth, do not ferment lactose, and show specific agglutination with standard antisera. The Flexner strains ferment mannite, the Shiga do not.

\* By F. A. Knott, M.D.

Sonne dysentery bacilli are not strict non lactose fermenters although the reaction is very delayed. Otherwise their reactions correspond closely to those of *Bact. dysenteriae* (Flexner). The Schmitz type dysentery bacilli resemble when judged by cultural reactions *Bact. dysenteriae* (Shiga) but they produce indole in broth a property usually found in non pathogens only. The organism known as the Newcastle bacillus, which can give rise to mild dysentery is an example of a further variation. In this case there may be slight motility and in those sugars fermented traces of gas may be formed. Evidently the atypical strains represent biological intermediates between the saprophytic *Bact. coli* and the highly pathogenic Flexner and Shiga strains.

Fortunately all these dysentery producing bacilli, typical and atypical, retain their antigenic individuality to a large extent, and, although in early subcultures they may be found in an magglutinable phase, later subcultures can be identified by clear-cut agglutination results with specific antisera. The Shiga strains all have the same antigenic make-up and usually a Shiga antiserum prepared against any one of them will agglutinate all the bacilli in that group. The position with regard to the Flexner organisms is more complicated. These organisms may carry any of at least four different antigenic components, usually named V, W, X and Z. If V is predominant the strain is termed V if Z is relatively more plentiful than the others it is the Z strain and so on. Bacilli containing equal amounts of all four components are described as belonging to the Y strain. There are, therefore five distinct serological Flexner types V, W, X, Z and Y. Antiserum specific for the first four are useful for exact identification such as may be necessary for epidemiological purposes. Y antiserum is adopted for diagnostic tests, because antibodies to all the components being present in quantity it will give reasonably high titre agglutination with nearly all Flexner strains. Boyd (1940) has recently carried this classification a stage further by showing that all Flexner strains carry a common group antigen, not included in the V-Z factors, and that if possession of this new common factor be taken as characteristic of a true Flexner dysentery strain three more members can be added. The newcomers are of interest in war-time owing to their particular distribution. Type 103 occurs in England, on the Continent the Near East, West Africa and India. Type 119 is unknown in Europe but present in the Far East and is common

in South Africa Type 88 is found in Europe and India and appears to be identical with the strain known in this country as the Newcastle bacillus Thus we have now three numbered types to add to those labelled V to Z Doubtless a more satisfactory nomenclature will soon be agreed upon. At the moment the important point is that these numbered types are, speaking clinically, true Flexner dysentery bacteria Certainly such strains, as well as the more atypical dysentery bacilli, particularly the Sonne variety, tend to have characteristic antigenic plans and to give rise to agglutinating antisera sufficiently specific for diagnostic purposes The method of quick direct slide agglutination of plate colonies by known antisera is very effective in this field.

There are in fact many points of similarity in the laboratory investigation of dysentery and the enteric fevers But there are also certain essential differences The latter become obvious when the pathology of the two infections is compared The short incubation period of dysentery is noticeable. There is no complicated invasion stage as in typhoid, with bacteræmia and passage of the bacilli to the liver and spleen before re-excretion *via* the bile into the intestines. Dysentery is at first a remarkably local disease. The highly toxic Shiga infections have a mechanism in many ways comparable to that seen in diphtheria The bacilli remain localised, producing acute necrosis of the intestinal mucosa, ulceration and bleeding From these areas the soluble dysentery exotoxin is rapidly absorbed and early in the infection gives rise to profound systemic effects The bacilli are plentiful in the faeces as soon as acute mucosal inflammation occurs.

Faecal cultures should be made as early as possible from either a fresh faecal specimen or a rectal swab Drying must not be allowed, if it is avoided dysentery bacilli remain alive at 15° C for several days. Repeated platings may be made upon MacConkey's medium or litmus-lactose-agar and any non-lactose fermenting colonies tested as to both their cultural and agglutination reactions. But if certain more recently devised selective media are available, their use is to be preferred, suppression of the ordinary saprophytic coliform bacteria making isolation of the dysentery colonies decidedly easier. Two devised by Wilson and Blair have been mentioned in connection with enteric fever (p. 271) The Emergency Public Health Laboratory service is already finding the second, namely the desoxycholate-citrate-agar, to be especially

useful in dysentery and definitely superior to the older media when only small numbers of the organisms are being passed. More recently Wilson and Blair (1941) have devised a third medium containing tellurite, iron and rosolic acid which they claim as specially favourable for isolating Flexner strains.

The organisms very rarely appear in either the blood or urine though they are often found *post mortem* in the mesocolic glands even in patients dying from other causes some time after recovery from dysentery (Fraenkel 1943). In the absence of a bacteraemic phase the toxic element is predominant but the patient does produce, though slowly anti bacterial antibodies (agglutinins) comparable to those met with in enteric fever but their titre in the serum is never very high. Moreover the dysentery bacilli, being devoid of flagella and therefore nonmotile, carry only O (somatic) agglutinogens. Thus agglutination takes place slowly and the tests must be allowed to remain at 57° C for at least six hours and at room temperature for some hours more before final readings are made. The serum should always be brought into contact with a fully representative series of dysentery strains owing to the variations in antigenic make-up already mentioned.

In dysentery it is rare to find an agglutination titre in the patient's serum higher than 1 in 250 and it is quite common for normal people to show a titre of 1 in 25 or 1 in 50 at times when the disease has recently been prevalent. In an active infection repeated tests usually show that the antibody content of the serum is rising. In carriers the agglutinins persist, but owing to the liability of normal serum to give positive reactions, only high titres should make one seriously consider a person as a likely carrier in the absence of demonstrable stool infection. At all stages of infection the most important diagnostic point is to obtain positive cultures from the faeces. Fortunately this is rather easier than in the case of enteric, a circumstance which offsets to some extent the slow agglutinin production and the uncertainty of agglutination results.

Agglutinins usually disappear quickly after convalescence, often within three months, in marked contrast to those resulting from enteric infection, which may persist for many years.

In most cases of active disease the bacilli cease to be excreted regularly in the stools within two or three weeks of convalescence. Only 2 to 5 per cent. of patients continue to void the organisms after three months. As in enteric, passing of the bacilli may be

very intermittent during the later stages, and it is most important to make repeated stool examinations before pronouncing a patient clear. Otherwise active carriers will certainly be released. It was noticeable during the last war that Shiga carriers were rarely missed because they remained toxic and looked ill. In such cases I found that numerous small superficial ulcers on an abnormally thick and red mucosa might be seen on sigmoidoscopy although the stools were formed (A.H.). Carriers of the other strains frequently show no signs or symptoms of any kind.

### **Prognosis.**

Shiga's bacillus generally gives rise to a more severe and protracted form of dysentery than Flexner's bacillus, and Sonne's bacillus generally causes nothing more than simple diarrhoea with some excess of mucus but no blood in the stools. With the treatment described below the mortality in dysentery epidemics is now rarely more than 2 per cent. Graham (1918) reported that in a large series of cases treated in the Toronto General Hospital at Salonica between June 1916 and May 1917 the mortality was less than 0.3 per cent. In two hundred severe cases, in which over twenty stools containing blood and mucus were passed per day and from which a dysentery bacillus was isolated, the death-rate was only 1 per cent. In the present war bacillary dysentery in the Middle East has been very mild, Shiga infections being hardly more severe than Flexner, and the mortality has been negligible.

### **Prophylaxis.**

All drinking water should be boiled when dysentery is prevalent, and no raw vegetables in the form of salads and no raw fruit unless it is carefully peeled should be eaten. Dysentery wards should be fly-screened, and the stools should be disinfected at once and destroyed as quickly as possible. A patient who has had dysentery should not be allowed to return to his unit until he can take a full Army ration without getting diarrhoea, and his faeces have contained no blood, mucus, dysentery bacilli or amoebic cysts for a fortnight.

The faeces of all troops should be passed into dust- and fly-proof receptacles containing cresol, and whenever possible they should be incinerated, failing this they should be deeply buried. In forward areas trench latrines should be dug at once and provided with well-fitting fly-proof covers. However thoroughly this is done, flies will continue to carry infection from the faeces of native carriers.

Camps and hospitals should therefore be situated as far away as possible from native quarters. Natives found defecating in the open near a camp should be punished, and latrines of the Moslem type efficiently fly proofed should be provided. Large and efficient fly traps should be kept at the entrance to cook houses, messes and canteens, all of which should be protected by fly proof netting. As in spite of these precautions flies are certain to enter all food should be kept covered. Special attention should be paid to the health of cooks, who should be instructed to wash their hands regularly, and no man should be allowed to have anything to do with the preparation of food if he is suffering from diarrhoea. Before leaving their wards, medical officers, nurses and orderlies who are looking after dysentery cases, should wash their hands with creosol.

Vaccination against bacillary dysentery has not been much employed owing to the very severe local and general reaction which follows the injection of killed cultures of the organism. The reaction is, however less severe if the bacilli are sensitised with antitoxin or treated for several days with 1 per cent formalin, and it is probable that in the future a considerable degree of protection will be obtained by the use of polyvalent vaccines, made whenever possible from strains isolated in the locality in which the vaccine is to be used. Gibson (1918) inoculated 1 150 men in the Near East and found that only 9 cases developed during the next fourteen weeks, whereas 66 cases appeared among 1 550 controls under similar conditions. Of the 9 cases in the first group 7 occurred within four weeks, and it is only after this interval that the maximum immunity occurs. The immunity lasts about six months, and the vaccine should therefore be given only at the beginning of the summer. It is usual to give two injections, with an interval of a week, of 1,500 and 3 000 million mixed Shiga and Flexner bacilli in equal numbers. It is important to realise that an epidemic of simple diarrhoea may be due to Sonne's bacillus, and although the symptoms may be so slight that the patients are ready to carry on, they should be isolated by being sent into hospital for a few days till the stools are no longer unformed.

In extensive epidemics it is difficult to deal with the carrier problem, but in small epidemics convalescent carriers should be kept under supervision until three negative examinations of the stools have been made at intervals of a week following the last

positive result. It seems likely that in the future the majority of dysentery carriers will be cured by chemotherapy. Thus Rantz and Kirby (1942) treated eleven carriers with 12 grm of sulphaguanidine daily for two to six days. In nine the bacilli disappeared from the stools during treatment and did not reappear in periods varying between one and three months. In two cases, however, the infection persisted in spite of the course of treatment being twice repeated.

### Treatment.

The patient should be kept warm in bed until the diarrhoea ceases and he should use a bed-pan. Though a completely fluid diet is generally recommended, I found in Salonica that the diet I used before the war and still use for acute enteritis and entero-colitis

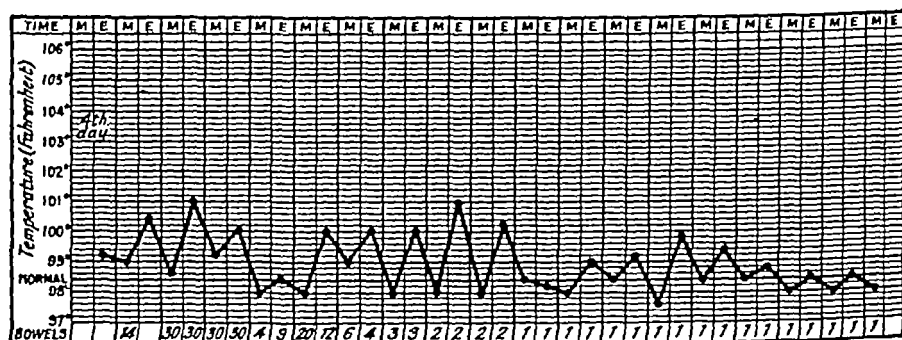


FIG 29—Bacillary dysentery treated with salts but no serum

was equally or more satisfactory. The patient is given arrow-root or cornflour made with water and sweetened with sugar. As soon as improvement occurs they are made with milk. Next custard, junket, purée of potato, and boiled rice, to which milk and sugar are added, are given, and the diet is very slowly increased. Even when the patient is convalescent he should avoid pips and skins of fruit and all raw vegetables, cooked vegetables being allowed only in the form of purées.

The intestinal lesions are not caused by the local action of the bacilli but by the action of their toxins, which are absorbed from the colon and cause necrosis of the mucous membrane on re-excretion. As the general symptoms, including such complications as nephritis and arthritis, are also due to the absorption of toxins, the usual treatment until recently was to delay their absorption and get rid of them from the bowel as rapidly and completely as

possible by the use of saline aperients, and to neutralise them after absorption by means of anti-dysenteric serum (Figs. 29 and 30). Now however it has become possible to prevent the formation of toxins by the destruction of the bacilli by chemotherapy, and the saline treatment which has been in use since the seventeenth century and was found of great value in the war of 1914-18 as well as in the American Civil War and the South African War has been given up.

In mild cases, including the majority of Sonne dysentery, recovery

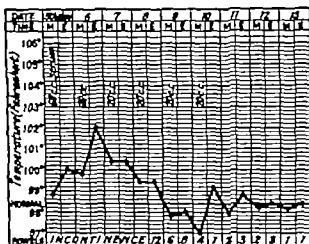


FIG. 30.—Bacillary dysentery treated with intravenous injections of serum.

is rapid with rest and a suitable diet, and chemotherapy and serotherapy are unnecessary.

**Serum treatment.**—A case of dysentery should be regarded as sufficiently severe for serum treatment if there is enough toxæmia to cause pyrexia and increased rapidity of the pulse, if a dozen or more stools are passed in twenty four hours or if the stools contain a large proportion of blood. A mild case which persists for more than three days also calls for serum treatment. If it is decided that serum is required it should be given at once without waiting for bacteriological confirmation of the diagnosis. The serum should be injected intravenously as the antitoxin is then immediately available to neutralise the toxin, whereas if subcutaneous injection is employed, a delay of some days occurs before the whole of the serum is absorbed. The strength of the concentrated serum now issued to the Army is 50 000 international units in less than

10 c.cm. An initial dose of 10 to 20 c cm. is sufficient even in fulminating cases

The Shiga toxin gives rise on injection into animals to a true neutralising antitoxin. The fact that all Shiga strains have the same antigenic structure is in favour of a high titre Shiga horse antiserum being therapeutically effective, and both statistical and clinical evidence support this. There are several war-time records showing that if Shiga antiserum be given early and in sufficient quantities, the severity of the symptoms, the duration of the disease and the mortality are all reduced. There is also some evidence that this serum can neutralise toxin even when already attached to the patient's cells, and therefore the object in its administration should be to saturate the tissues at once with antitoxin by injecting a large dose by the intravenous route

To be fully effective against the Flexner strains an antiserum should possess anti-bacterial as well as anti-toxic power. For this reason Flexner antiserum is prepared by injecting the horse with the bacilli themselves and not toxin alone. Most of the cases successfully treated by antiserum have received a product obtained by injecting the animal first with Shiga toxin and then with both Shiga and Flexner bacillary bodies. The results are best if local strains are used in the preparation of the serum

In asthmatics and in patients, who have on any previous occasion received dysentery, diphtheria or tetanus antitoxin or other serum, the danger of anaphylaxis can be overcome by a preliminary subcutaneous injection of  $\frac{1}{10}$  c cm of the serum half an hour before the first large injection. The risk, however, has been greatly reduced by the use of proteolytic enzymes in preparing the serum, and it is now never necessary to substitute intramuscular for intravenous injections (Poole, 1942). Formerly, however, the danger of anaphylaxis was not merely theoretical. In July 1916 a man died on a hospital ship a few minutes after 20 c cm of anti-dysenteric serum had been injected intravenously, he had had three previous injections without ill effect, the last having been at Salonica twenty-four days earlier. He felt "funny" and became pale during the injection, his whole body then became deeply flushed. In a few minutes the flushing was replaced by extreme cyanosis, his pulse became weaker and he died in about a quarter of an hour. As anaphylactic shock can be completely overcome by adrenaline, a syringe filled with a 1 in 1000 solution should always be at hand

when serum is injected. Five minims of adrenaline can be slowly injected at once and thereafter one minim every minute until all symptoms have disappeared even if they last for an hour or more.

*Chemotherapy* — Sulphaguanidine (p-amino-N-guanyl benzene-sulphonamide) and sulphapyridine have given promising results in the treatment of bacillary dysentery. The former was chosen for trial by Marshall Bratton White and Litchfield (1940) on account of its solubility in water and limited absorption from the intestine in addition to its strong bactericidal powers. Owing to its limited absorption it acts within the intestine in a relatively high concentration and is therefore likely to be effective in infections which are chiefly localised to the bowel.

Anderson and Cruckshank (1941) treated 41 adults in L.C.O. hospitals suffering from acute Flexner dysentery with 3 gm. of sulphaguanidine three times a day for two days followed by 2 gm. twice a day for two to five days with excellent results. The percentage who remained carriers during convalescence was half of that among control cases in the same epidemic who received no chemotherapy. Hardy, Watt, Peterson and Schlosser (1942) found that 45 carriers of the Flexner and Newcastle organism were quickly cured by treatment with sulphaguanidine.

The treatment of bacillary dysentery with sulphaguanidine in the Middle East has been described by Boyd and Hamilton Fairley (1943). Within twenty four to forty-eight hours the patient feels better abdominal pain and tenesmus are relieved, and the diarrhoea rapidly subsides so that within five or six days the bowels are only acting once or twice in the twenty four hours. Blood quickly disappears from the stools and the quantity of mucus is reduced, though its complete disappearance is comparatively slow. The temperature and pulse rate fall frequently reaching normal in one to three days. The dose is 2 gm. every four hours for three days, followed by 2 gm. every eight hours for four days. Treatment should be begun directly a clinical diagnosis has been made without waiting for bacteriological confirmation. Cases treated within twenty four to thirty-six hours of the onset recover in a dramatic fashion. Subacute and chronic cases respond well, healing being generally complete within a fortnight.

The action of sulphaguanidine being bactericidal, it prevents further damage to the bowel, and the production of toxin is at once diminished with consequent rapid improvement in the general

symptoms It has no effect on the dysentery exotoxin already circulating in the blood, so this must be neutralised by injection of serum in severe cases Only mild toxic symptoms have been produced by the drug These are headache, malaise, vomiting, and occasional transient erythematous or red papular rashes Hæmaturia and anuria, agranulocytosis, hæmolytic anaemia, and jaundice were not observed in any of their series of over 500 cases, but slight and transient hæmaturia and pain occurred in one case recorded by Bulmer and Priest (1942)

Paulley (1942), as a result of observations on strictly comparable groups of Air Force personnel suffering from dysentery in the Middle East, conclude that sulphapyridine is preferable to sulphaguanidine In the first series of 73 cases of mild and moderate severity sulphapyridine used from the onset was not followed by a single failure, and clinical improvement always occurred within one or two days In no case was blood passed for more than three days On the other hand, of 52 cases receiving the usually prescribed dosage of sulphaguanidine 5 failed to show satisfactory response In a Shiga epidemic the figures were even more conclusive, 5 out of 13 sulphaguanidine cases fared worse than any sulphapyridine case From published work and from observation of other cases treated in hospitals in the Middle East Paulley concluded that though sulphaguanidine is often as effective as sulphapyridine, cases frequently occur in which it appears to be inferior He suggested that this might be due to one or more of the following causes (1) the dosage of sulphaguanidine was not high enough, (2) sulphapyridine has a greater bacteriostatic action than sulphaguanidine *in vivo*, (3) the attainment of a high bowel lumen concentration may after all not be as essential as the attainment of a high blood concentration in dysentery Sulphapyridine has the further advantage over sulphaguanidine of being more plentiful and cheaper, and a smaller dose is required for each case The greater risk of agranulocytosis is of little importance, as the drug very rarely need be given for more than four days It has, however, the disadvantage of causing anorexia, nausea and depression much more frequently than sulphaguanidine

When the diarrhoea is excessive, dehydration can be overcome and collapse prevented by injecting normal saline solution into a vein by the drip method, or by drip transfusion if the patient is anæmic from loss of blood This is of great importance when

sulphonamides are given, as otherwise crystallisation might occur in the renal tubules with production of anuria

When the patient is kept awake by abdominal pain, and when diarrhoea persists during the night and is leading to exhaustion  $\frac{1}{4}$  gr of morphia may be injected with  $\frac{1}{16}$  gr of atropine sulphate at 10 p.m. I found in Salonica that charcoal was very effective in diminishing the frequency of defecation and in deodorising offensive watery stools. At the same time flatulence, which is often excessive and is the chief cause of colicky pain, is greatly reduced. Half an ounce of charcoal to a teaspoonful of sweetened arrowroot makes a palatable food, it should be given three times a day in the more acute stages

Normal stools never contain *Bact. dysenteriae* so a patient should be kept in hospital till his stools not only give no dysentery organism on culture but have also been of normal consistence and have contained no mucus for five days. When diarrhoea persists, sigmoidoscopy always shows that healing is incomplete.

### AMOEBIĆ DYSENTERY

Amoebic dysentery is endemic in many tropical and subtropical countries and is especially common in districts where the sanitation is deficient. It has sometimes been stated that it never occurs in epidemic form. The error of this was finally established by the severe Chicago epidemic of 1933 when over 1 000 cases with 60 deaths occurred as a result of pollution by sewage of the water supply to an hotel. Small epidemics were frequent among American soldiers during the Philippine insurrection in 1905. An extensive epidemic developed in the summer of 1915 in the British Army at Gallipoli. A large proportion of the thousand sick men who were daily removed from the Peninsula during August and September had dysentery and during those months amoebic was at least as common as the bacillary form. The incidence was smaller in October and the cold and rain in the great gale at the end of November were quickly followed by its disappearance. Amoebic dysentery was brought to Gallipoli from Egypt where Wenyon and O'Connor (1917) found that 13.5% of healthy natives were carriers. It was common in Mesopotamia, but rare in Salonica, where it accounted for less than 3% of the total cases of dysentery

Amoebic dysentery first appeared in France in September 1915,

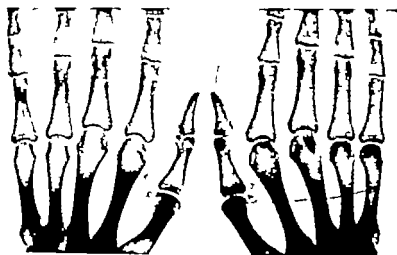
and though it remained rare, the number of cases steadily increased among both British and French soldiers. The infection was introduced by carriers from India, Morocco and Senegal, and the arrival of troops from Gallipoli led to a fresh source of infection in 1916, the disease finally spreading to men who had never seen service out of France. In the stools of soldiers sent from France as convalescent from dysentery cysts of *E. histolytica* were present in about 10 per cent. of the English and 20 per cent. of the Australian cases, although only one-third had previously been in a country where amoebic dysentery is endemic (Inman and Lille, 1917). There was no amoebic dysentery among British soldiers in France in 1939 and 1940, but it has been very prevalent in the Army in the Middle East, frequently in association with infection with Flexner's and Shiga's bacillus. About 25 per cent. of cases admitted into hospital for dysentery are amoebic, but the proportion is higher for recurrent and chronic cases.

### **The *Entamoeba histolytica*.**

Parasitic amoebæ—entamoebæ—are distinct from the non-parasitic amoebæ of the soil and water in their morphological characters. They become parasites in the intestines when introduced by mouth, whereas non-parasitic amoebæ disappear spontaneously from the faeces within a week of being swallowed. There are five varieties of entamoebæ (Fig 31, Plate II). Only one, the *E. histolytica*, is pathogenic, the others, *E. coli*, *Endolimax nana*, *Iodamoeba bütschlii* and *Dientamoeba fragilis*, being harmless parasites found in the stools of many normal people.

The *E. histolytica*, when given to kittens, puppies and monkeys by mouth, produces characteristic ulcers in the colon and abscesses in the liver, which contain the amoebæ but no bacteria, whereas the non-pathogenic amoebæ produce no lesion.

The *E. histolytica* has three stages in its life history—a motile active form, an immobile cystic form, and an intermediate precystic form. The motile form is found in fluid and semi-fluid stools, the cystic form in semi-formed and formed stools, and the precystic form in both fluid and formed stools. The active form of the *E. histolytica* varies in size from 15 to 60  $\mu$  and is larger than any of the other entamoebæ except the *E. coli*, its movements are more active and progressive, and alone among the entamoebæ it frequently contains red blood corpuscles (Fig 31, 1 (a) and 6). The cystic form is spherical with a diameter of 6 to 20  $\mu$ , and it



110 11A.

Radiograph shows no increased transparency of left hand, taken immediately after recovery from hysterical paralysis of sixteen months' duration.

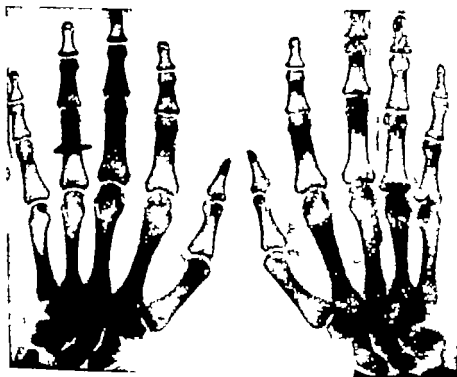


FIG 11B.

Radiograph of right and left hands, showing increased transparency of the bones of the right 4th and 5th fingers and to some extent the 3rd finger. Taken after recovery from contracture involving the 4th and 5th fingers. 11th partial contracture of the middle finger, which had persisted for 20 months after onset of the hand. The little and ring finger also show atrophy of the soft parts. (Major J. C. Ven. 1894.)

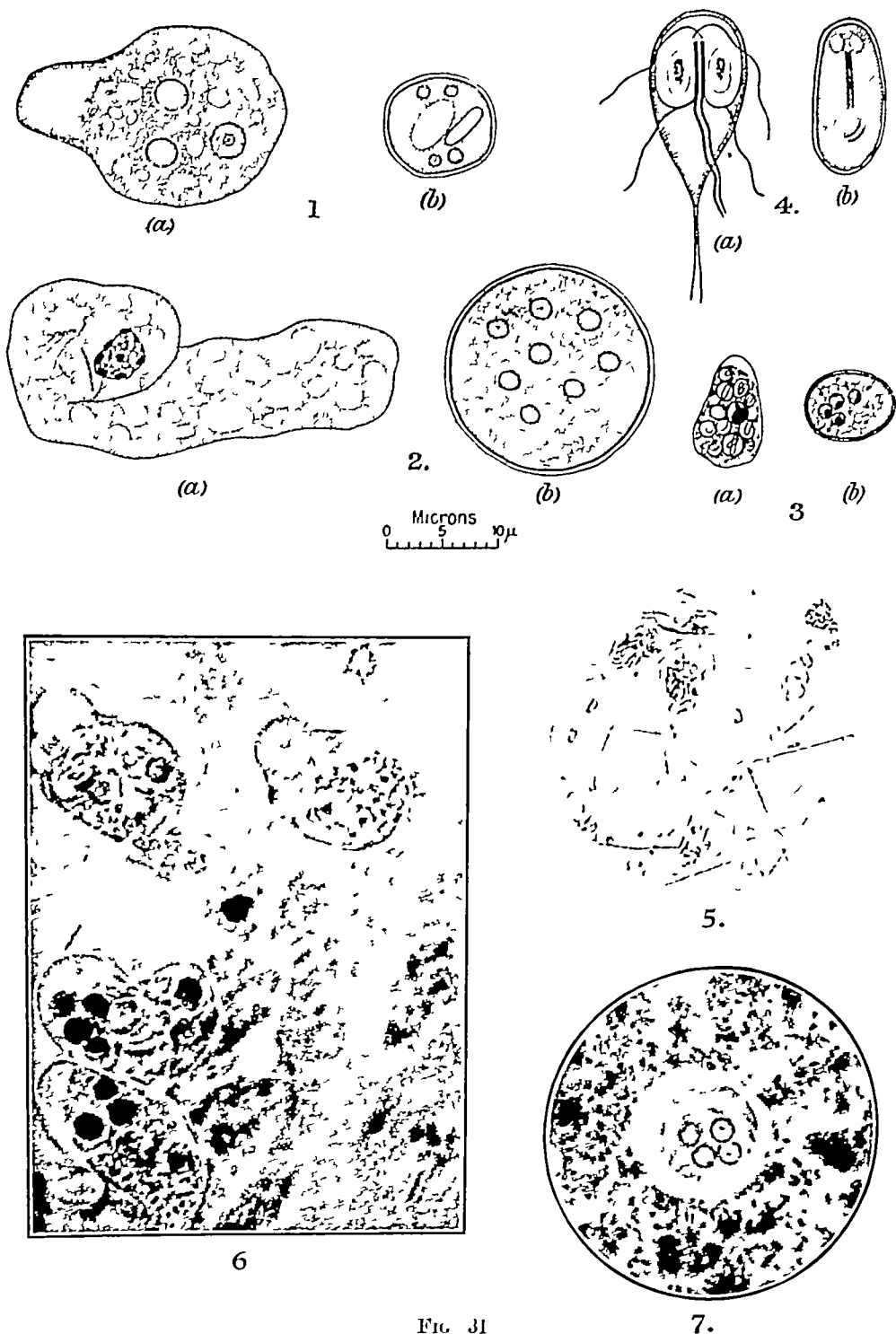


FIG. 31

7.

- 1 *Entamoeba histolytica* (a) large tissue invading form with ectoplasmic pseudopodia and containing two red blood corpuscles (b) encysted form with four nuclei chromidial body and vacuoli (C. M. Wenyon)
- 2 *Entamoeba coli* (a) large entamoeba, (b) encysted form (C. M. Wenyon)
- 3 *Endolimax nana* (a) showing nuclei and numerous vacuoles containing ingested bacteria, (b) quadrinucleate cyst (Clifford Dobell)
- 4 *Lambia intestinalis* (a) surface view showing sucking disc, two nuclei and eight flagella, (b) encysted form with two nuclei (C. M. Wenyon)
- 5 Photomicrograph showing *Lambia* and cholesterol crystals in gall bladder contents, obtained by biliary drainage ( $\times 1200$ ) (B. B. Vincent Lyon)
- 6 Unstained microphotograph of stool showing *Entamoeba histolytica*, three of its corpuscles ( $\times 750$ ) (Colonel C. J. Craig)
- 7 Cyst of *E. histolytica*, with four nuclei, stained (C. J. Craig)

contains 1 2 or 4 nuclei (Fig 31, 1 (b) and 7) An expert protozoologist has little difficulty in distinguishing both the active and cystic stages of the *E histolytica* from those of the four non pathogenic entamoebæ but mistakes are frequently made by the inexperienced.

The active form of *E histolytica* dies too quickly outside the body to take any part in spreading the disease. Amoebic cysts, however survive many days and even weeks in moist faeces and in water The infection can consequently be conveyed through contamination of a water supply or vegetable garden, contamination of food by the hands of carriers, and of food by the excreta of flies. As the cysts are rapidly killed by drying they cannot be carried in dust.

Wenyon and O Connor's investigations showed that house-flies are much the most important means of spreading dysentery in Egypt. Their two favourite articles of diet are faeces and jam. Though they begin to excrete what they have ingested within half an hour amoebic cysts (and also *Bact. dysenteriae*) have been found in their intestines and excreta up to twenty four hours after they had fed on infected stools. A considerable number caught by Wenyon and O Connor in different parts of Alexandria, including one caught in a cook house, deposited faeces containing *E histolytica* and *Bact coli*. As a fly generally defecates whenever it eats, it leaves a deposit containing amoebic cysts (or dysenteric bacilli) on jam and other human food upon which it settles, if it has had access to infected faeces within the preceding twenty four hours. The infection is probably never carried by the fly on its legs, body or proboscis, as it cleans itself after leaving the faeces, and the remaining traces dry so rapidly that no cysts or bacteria could survive long enough to be deposited on food.

The stools of convalescent and apparently healthy carriers are a more fertile source of infection than those of patients still suffering from dysentery as their formed or semi formed stools contain many more cysts than the fluid or semi-fluid stools passed during an attack of dysentery Among 1979 healthy British troops, whose stools were examined by Wenyon and O Connor in Alexandria during 1916 106 (5.3 per cent.) were found to be carriers of cysts of *E histolytica* although only 16 of them had had attacks of diarrhoea or abdominal pain. Fatal dysentery can be produced in cats by feeding them on the cyst-containing faeces of symptom

free carriers Even when provided with latrines the average native in the Middle East prefers to deposit his excreta wherever he happens to be when the desire to defæcate seizes him D. and J. G. Thomson examined many samples of fæces collected near Alexandria from the foreshore and the sandy stretches between the sea-front promenade and the town; a high percentage contained cysts of *E. histolytica* as well as bacteria of the typhoid-dysentery group

So-called healthy carriers, who have no symptoms but are passing amœbic cysts, always have an intestinal lesion This may be nothing more than minute areas of necrosis of the mucous membrane, which may only be recognisable on microscopical examination (Craig) More commonly typical amœbic ulcers, generally very minute, but occasionally quite large, are present, especially in the cæcum and ascending colon Carriers suffering from simple diarrhœa have actual ulcers, which are as large and numerous as those found in patients with typical dysenteric symptoms

The wall of the amœbic cyst is unaffected by the gastric juice, but is digested by the pancreatic juice, so that the amœbæ are set free in the intestines Amœbæ liberated from a two- or four-nucleated cyst divide once or twice to form two or four active amœbæ, which burrow from the lumen of the colon through the mucous membrane into the submucous tissue The active amœbæ continue to multiply by simple division both in the lumen of the bowel and in the tissues The division continues indefinitely in the tissues, but ceases in the lumen of the bowel when the conditions become unfavourable The active amœbæ then assume a rounded pre-cystic form, which secretes a transparent shell and becomes a cyst. The single nucleus of the encysted amœbæ generally divides once or twice, a fully developed cyst having one or more frequently two or four nuclei Cysts are never found in the tissues and never form active amœbæ outside the body

### Pathology.

Sigmoidoscopic observations have added precision to our knowledge of the morbid anatomy of amœbic dysentery, as every stage in the natural history of the disease can be observed without the alterations seen after death as a result of the absence of circulating blood and the presence of *post mortem* degeneration I made numerous observations of this kind at Lemnos in 1916 and a smaller number since then in England, and similar observations have been published by Manson-Bahr.

The proximal part of the colon is first involved, and even when the whole colon later becomes affected the disease is generally most advanced in the cæcum. The appendix is sometimes affected, but the ileum is almost always spared.

Amoebæ which have reached the submucous tissue of the colon, continue to multiply and destroy the surrounding cells by means of the cytolyzins they excrete until a pocket containing amoebæ necrotic tissue and mucous forma. This lifts the mucous membrane covering it above the level of its surroundings. The raised mucous membrane becomes red, and owing to deficient circulation its centre undergoes necrosis, so that a yellowish spot is seen in the centre of a small rounded red swelling which is sharply defined from the surrounding normal pink mucous membrane. The appearance is very similar to that of a small boil on the healthy skin. The necrotic tissue soon disappears and leaves a minute hole connecting the submucous pocket with the intestinal lumen. The lesion grows to form a flask-shaped cavity and then a spreading ulcer with an overhanging margin and infiltrated submucous or muscular tissue as its base. Later the ulcers coalesce to form large irregular ulcerated areas. Others communicate with each other by submucous channels. This may result in the separation of black sloughs of necrosed mucous membrane which may be several inches in length. These are passed in the stools or are found after death attached to the edge of large ulcers. In these cases the amoebæ reach the subserous tissue, and the peritoneum may be covered with purulent fibrinous patches. Even in the severest cases areas of normal mucous membrane remain, especially in the distal half of the colon.

It is generally said that on healing pigmented scars remain. This describes what is found on *post mortem* examination of chronic cases, but I have frequently watched the effect of emetine with the sigmoidoscope and seen the complete disappearance of even large ulcers, no trace of a scar or pigmentation being visible after ten to fourteen days.

Microscopically there is a remarkable absence of round cell infiltration in the early stages before secondary infection has taken place, but secondary bacterial infection with cellular reaction is generally present in all but very early lesions. Amoebæ are seen in the necrotic areas of the mucous, submucous and muscular coats within and around the crypts of Lieberkühn and within the capil

laries, venules and lymph spaces They may be carried from the submucous veins to the liver, where they give rise to inflammation and suppuration (*vide* p 311).

### Symptoms.

The incubation period is often short, but is sometimes prolonged Crean records the case of a soldier who landed in Alexandria from England at 4 30 p.m. on a Friday He had never been abroad before. He at once marched to Mustapha, where there were several cases of amœbic dysentery. At 9 a.m. on Sunday—forty hours after his arrival—he passed blood and mucus, which contained amœbæ

The onset of amœbic dysentery may be acute, but in a considerable proportion of cases the disease begins as chronic diarrhœa or as diarrhœa alternating with constipation In acute cases ten to twenty or even more stools are passed a day When the disease has fully developed, blood and mucus are almost always present in the stools, which consist of a few drachms of greenish yellow or dirty brown mucus, or a grey muco-purulent mass, suspended in semi-fæculent or serous fluid, which is often blood-stained, the blood is either intimately mixed with the stool or is present in streaks In other cases large separate fragments of mucus stained bright red are seen

If a microscopical examination is made within half an hour of the passage of a stool, active amœbæ can generally be found without difficulty in the blood-stained mucus, but they may also be present in mucus without obvious blood and in watery stools free from blood and mucus The amœbæ are generally scattered among numerous isolated nuclei, but few intact pus cells, and often numerous red corpuscles

Gripping pains, which are worst shortly before and during defæcation, are always present to a greater or less extent across the lower part of the abdomen Tenesmus, the painful bearing down and straining sensation which occurs during and for some time after defæcation, is common, but it is severe only if the rectum is ulcerated

The thickened colon can often be felt, especially in the right and to a less extent in the left iliac fossa, as a very tender tumour, when the cæcum is chiefly involved, appendicitis may be simulated, but the tenderness is found to extend over the whole cæcum and ascending colon instead of being confined to the appendix itself

Constitutional symptoms are generally slight and apart from complications fever is either completely absent or low and intermittent even in acute cases. Leucocytosis is generally present, and there is a relative increase in the mononuclear cells in severe cases the number of leucocytes per c.mm. often exceeds 20 000 and sometimes even 30 000.

In the fulminating gangrenous type of amoebic dysentery the patient passes very numerous stools, which often consist of blood with a little mucus but no faeces. When extensive gangrene occurs, the colon may become paralysed, and only a few stools containing black sloughs are passed. Abdominal pain is severe, and palpation shows that parts of the colon are greatly thickened and extremely tender. In such cases perforation and general or local peritonitis may occur.

Mild and unrecognised amoebic dysentery is common and may be the precursor of hepatic abscess. A history of slight diarrhoea without the passage of blood often alternating with constipation and sometimes accompanied by pain in the right side of the abdomen, is obtained. The comparatively mild symptoms are probably due to the lesion being confined to the caecum and ascending colon which may be tender and slightly thickened. In such cases amoebae are difficult to find in the stools, unless frequent search is made for a little blood-stained mucus. The diarrhoea, which was common among the troops at Gallipoli, was probably in many instances a mild form of amoebic dysentery. The disease may even be completely latent, ulceration being found *post mortem* in patients dying from amoebic abscess of the liver or from some intercurrent disease.

In chronic amoebic dysentery which often develops in spite of efficient treatment, amoebic cysts are passed and symptoms may continue for months or years often with long intermissions. emaciation, anaemia, and general asthenia result, and the patient ultimately dies from exhaustion or hepatic complications.

Several cases of sciatic neuritis and a smaller number of neuritis affecting other nerves were observed in association with the outbreak of amoebic dysentery in the Mediterranean Forces during the last war.

### Diagnosis

In any neighbourhood in which the disease is known to occur every case of diarrhoea should be regarded as a possible case of dysentery and if blood or mucus or both are passed or if tenesmus

is well marked, it should be regarded as a probable case of dysentery. Other causes of similar symptoms, the most important of which is a growth of the pelvic colon or rectum, should not be forgotten. I found an inoperable growth in the rectum of a governess sent home from Persia for what was supposed to be chronic dysentery, and I saw a case in Lemnos, in which a man with a similar growth had been treated for some weeks with emetine. A careful inquiry into the history, a rectal examination, and in doubtful cases sigmoidoscopy should prevent such a mistake being made.

If symptoms of appendicitis occur in a patient, in whom there is a possibility that amœbic dysentery is present, the stools should be examined for amœbæ, and the effect of emetine injections should be tried before advising operation, except in severe cases, in which the delay caused by failure of the treatment might prove dangerous. The majority of deaths in the Chicago epidemic of amœbic dysentery were in patients operated upon for supposed appendicitis.

For the clinical differentiation of amœbic and bacillary dysentery, see p. 289. Although an amœba with refractile ectoplasm clearly differentiated from the granular or vacuolated endoplasm, an indistinct nucleus, and active movement is generally the *E. histolytica*, its characteristics vary so much that it may be quite indistinguishable from the *E. coli*, in which the distinction between ectoplasm and endoplasm is not present, the nuclei are distinct, and the movements are very sluggish. The inclusion of red corpuscles is, however, conclusive evidence in favour of *E. histolytica*, and no acute case of dysentery should be diagnosed as amœbic unless amœbæ containing red corpuscles are found, as they are almost invariably present.

When for any reason it is impossible to make an immediate microscopical examination of the stools, treatment with emetine should at once be instituted in suspicious cases arising in a district in which the disease is known to have occurred, rapid improvement can be regarded as almost conclusive proof of the amœbic origin of the dysentery.

### Prognosis.

If emetine is promptly given, death should hardly ever occur. When treatment is delayed, the prognosis is less good and the actual dysentery is likely to be followed by a non-specific chronic colitis, which may persist for many months. Incontinence of fæces,

persistent vomiting, hiccup and the passage of sloughs are the most serious symptoms.

The amoebic dysentery at Gallipoli 'lowered vitality more than it destroyed' it was the universal sickness, not the occasional death that mattered.' It was, in fact, one of the deciding factors in the failure of the campaign.

### Prophylaxis

The prophylaxis of amoebic dysentery is in general the same as that of bacillary dysentery (*vide* p 294). Neither chlorination nor the addition of acid sodium sulphate tablets to water destroys encysted amoebae. All drinking water should therefore be boiled when amoebic dysentery is prevalent and no raw vegetables in the form of salads and no raw fruit unless it is carefully peeled should be eaten. A patient who has had dysentery should not be allowed to return to his unit until he can take a full Army ration without getting diarrhoea, and his faeces have contained no blood, mucus, or amoebic cysts for a fortnight. He should still be given chiniofon after his apparent recovery as described in the section on treatment. Before leaving their wards, medical officers, nurses and orderlies, who are looking after dysentery cases, should wash their hands with cresol (1 in 10) which Wenyon and O'Connor have shown is the best antiseptic for destroying amoebic cysts.

### Treatment

The general and dietetic treatment is the same as for bacillary dysentery. Craig advises complete abstinence from alcohol in even the mildest cases of amoebic dysentery as he believes that there is "no more potent cause of acute diarrhoeal or dysenteric symptoms in any infection with *E. histolytica* than indulgence in alcoholic beverages.

Directly the diagnosis has been made by the discovery of the *Entamoeba histolytica* in the stools, gr 1 of emetine hydrochloride should be injected subcutaneously every day for the next twelve days. In very severe cases gr  $\frac{1}{2}$  to 1 dissolved in 5 c.c. of normal saline solution should be injected intravenously. The amoebae, blood and mucus generally disappear from the stools between one and six days after the first injection. In the rare cases of amoebic dysentery in which emetine fails more or less completely a mixed infection is certainly present, and antidyenteric serum should be given in addition to the emetine. It is unnecessary to persevere with emetine until the stools are solid, as after the blood and mucus

have disappeared the stools may remain loose for a time owing to the post-dysenteric and non-specific colitis still present. They sometimes become rapidly solid on discontinuing the emetine, which may act as a mild intestinal irritant. If any blood or mucus reappears in the stools, gr. 1 of emetine hydrochloride should be injected every night for six nights, but not until at least three weeks have elapsed since the termination of the initial course of treatment. It is unusual for more than one such relapse to occur. In the very exceptional cases in which emetine injections cause vomiting, this may be prevented by the simultaneous injection of a small dose of morphia.

Dale found that repeated injections of emetine in cats had a cumulative action upon the heart. In very exceptional cases emetine has seemed to cause shortness of breath and irregularity of the pulse in patients who were not kept in bed, but these symptoms always disappeared after a few days' rest. Pain and stiffness of the legs have also been said to follow the use of emetine, but this is only temporary, and there is no evidence that genuine neuritis ever occurs.

As amœbic cysts are often still present in the fæces of patients who have apparently recovered from an attack of dysentery, it has been supposed that active amœbæ may develop from the cysts and cause a recurrence of symptoms if the patient's general health is depressed or his colon is irritated, and that a chronic infection of this sort may give rise to amœbic hepatitis or abscess of the liver. Wenyon and O'Connor have, however, shown that this is not the case, as cysts do not liberate active amœbæ anywhere but in the small intestine. But the presence of cysts in the fæces indicates that free amœbæ must be present in the colon, and it is these that are a danger to the patient. From the patient's point of view it is the cyst-forming amœbæ rather than the cysts themselves which require destruction, just as it is the ankylostoma which produces the ova found in the stools in cases of ankylostomiasis, and not the ova themselves which must be destroyed. It is therefore of comparatively small importance that amœbic cysts are very resistant to emetine, as if the drug is properly administered the amœbæ which produce the cysts are destroyed and the cysts disappear from the fæces. The frequently expressed view that some cases of infection with *E. histolytica* do not respond to treatment because only the resistant cysts are present in the intestine

is thus incorrect. But the cysts present in the faeces *after* they have been passed must be destroyed in order to prevent the spread of the disease.

It is often impossible to eradicate the infection in chronic carriers by means of emetine injections. The cyst-producing amoebae appear to be shut off from the circulation and are therefore more readily destroyed by a drug given by mouth. The most satisfactory treatment for the residual infection following an acute or subacute attack is by means of an amoebicidal drug which does not interfere with the patient's daily occupation. The best of these is chiniofon, (iod-oxy-quinolme-sulphonic acid) which contains 28 per cent of iodine and was first introduced under the trade name of yatren and is also known as quinoxyl and anayodin. Three 4-grain pills are given three times a day for eight days. If amoebae or cysts are still present in the stools the treatment is repeated after an interval of a week. The stools are then examined monthly for six months and the treatment is repeated if amoebae or cysts are discovered. Craig eliminated the infection in 90 per cent. of carriers and patients with mild infection after a single course of treatment and in the remaining 10 per cent. after repeated courses. Chiniofon is no more effective when given by rectum, as often recommended, than by mouth (Craig). Carbarsone, which contains 28 per cent. of arsenic, is less effective than chiniofon and occasionally gives rise to serious symptoms of arsenical poisoning. Emetine-bismuth iodide, which has been much used since the last war by British physicians, produces a good deal of gastro-intestinal irritation and should be replaced by chiniofon. With the latter it is unnecessary to restrict either diet or exercise even if slight diarrhoea occurs (Craig).

In order to minimise the risk of recurrence a course of treatment with chiniofon should be given to every patient with amoebic dysentery after the diarrhoea has been controlled by the use of emetine.

#### AMOEBO HEPATITIS AND HEPATIC ABSCESS

Considering the large number of cases of amoebic dysentery which occurred in the Mediterranean Expeditionary Force in the last war remarkably few soldiers developed acute or subacute hepatitis and hepatic abscesses, probably owing to the thorough treatment with emetine which the majority received. But the

treatment was often not continued for a sufficient period, and many slight cases escaped recognition altogether, so that in the ten years following the war a good many cases of subacute hepatitis and hepatic abscess developed in men who had served in the East and chronic amœbic hepatitis became comparatively common. I saw cases of both kinds as long as fifteen years after the original infection had been contracted in Egypt or Gallipoli. In a case seen in 1941 the abscess developed twenty-two years after the original infection in Mesopotamia, where the patient was thoroughly treated for it in 1919. He had had occasional slight attacks of diarrhœa lasting a few days, sometimes with traces of blood in the stools, but there had been none for several months before the abscess was discovered.

It is unlikely that acute and subacute amœbic hepatitis and hepatic abscess will be any more common in the Army in North Africa in this war than the last, but it is important to be familiar with the symptoms of these diseases, as they may be the cause of obscure illness in men who give no clear history of having had dysentery, and chronic amœbic hepatitis, the frequency of which is still insufficiently recognised, is likely to be common for many years to come among men who have served in the East.

### **Ætiology.**

Amœbic hepatitis and amœbic abscess of the liver are invariably secondary to amœbic dysentery. In 98 per cent of fatal cases Rogers either obtained a history of dysentery or found amœbic ulcers or scars of ulcers in the large intestine. The patient had had dysentery in the past or was still suffering from it in about 75 per cent of acute and subacute cases, and in an additional 15 per cent. there was a history of diarrhœa, which was doubtless a mild form of amœbic dysentery. In the remaining cases amœbic ulcers had probably been present in the cæcum or ascending colon, situations in which they do not necessarily give rise to any symptoms. When hepatitis develops during an attack of dysentery, the latter generally becomes less severe as the inflammation in the liver progresses.

The geographical distribution of the disease is identical with that of amœbic dysentery, but as it may develop in chronic cases and long after the onset of dysentery, it is not uncommon, especially in the chronic form, among individuals who have returned to

England from the East. Chronic alcoholism is an important predisposing cause.

### Pathology

The amœbæ which collect in the thrombosed veins at the base of dysenteric ulcers, pass to the liver by the portal vein. Nothing more is known as to the pathology of non-suppurative amœbic hepatitis, as death does not occur unless an abscess forms, but the hepatic insufficiency which is always found by the leucose test to be present, indicates that the infection is widespread and uniform through the liver. An abscess forms if a local accumulation of amœbæ gives rise to thrombosis in a portal radicle. The circulation is obstructed and necrosis of the surrounding tissue occurs at the same time pus is secreted as a result of the irritant action of the amœbæ on the liver tissue. The wall of the abscess is at first formed by necrotic liver tissue, but the cavity gradually becomes limited by a fibrous capsule produced by the inflammatory reaction. In 70 per cent. of cases a single abscess is present in nearly half of the others there are two abscesses, in a quarter there are three, and in the remainder there are four or more. A single abscess is found five times as often in the right as in the left lobe of the liver.

The contents of the abscess consist of necrosed liver cells, leucocytes, red corpuscles fat droplets and occasionally amœbæ but no bacteria. In chronic cases all the amœbæ may have died but they can still be found in the material obtained by scraping the wall of the abscess. In rare cases an amœbic abscess becomes spontaneously sterile, and its dried remains have been found at a *post mortem* examination years afterwards. When an hepatic abscess reaches the surface, adhesions form between the liver and the adjoining structures, so that the contents of the abscess may burst into the lung stomach or bowel, or open externally without infecting the serous cavities.

### Acute and Subacute Amœbic Hepatitis and Hepatic Abscess

#### Symptoms

About one-tenth of the cases of acute amœbic hepatitis occurring in India are of a fulminating type, in which the whole liver is riddled with small collections of pus with no fibrous tissue separating them from the surrounding liver substance—a condition which

rarely, if ever, occurs outside the endemic areas. There is always a definite history of dysentery, which is often still present when the hepatitis develops. The liver rapidly increases in size, it is very painful and extremely tender. Slight jaundice may be present. The temperature is high with rapid remissions, the rise being often accompanied by rigors and the fall by copious sweating. Leucocytosis is well marked. Death generally occurs between 6 and 18 days from the onset of symptoms.

As most cases of subacute amœbic hepatitis subside completely with injections of emetine, it is clear that in the early stages no suppuration is present. Discomfort and a sense of weight are felt in the right hypochondrium in the slighter cases, but in the more severe ones the pain may be so great that the patient is hardly able to move, and he is often unable to lie on his left side owing to the dragging pain caused by the change of position. Pain may also be referred to the right shoulder and occasionally to the right arm, especially when the upper part of the liver is involved. The liver is enlarged and tender, but the rigidity of the abdominal muscles may be so great that it is impossible to feel its edge. Pressure exerted on the lower part of the right thorax, with one hand behind and the other in front, gives rise to pain. Slight jaundice is occasionally present in the severer cases. The appetite is impaired or lost, and the patient rapidly becomes weak and emaciated.

The temperature is generally remittent, varying between  $100^{\circ}$  in the morning and  $103^{\circ}$  or  $104^{\circ}$  in the evening. In severe cases it remains high with only small remissions, but in more chronic cases, especially when the abscess bulges through the capsule of the liver and the tension within its cavity consequently falls, the fever is less marked, it may then be low, continued or relapsing, and may finally disappear. Copious sweating is common in the more severe cases. Moderate leucocytosis is always present, and the proportion of polymorphonuclear cells is considerably increased, especially when actual suppuration has occurred, if the percentage is less than 70, simple hepatitis, and not an abscess, is probably present.

The upper part of the liver is most frequently involved, and the main increase in dullness is then in the upward direction, but the lower border is also abnormally low. The lower ribs bulge, and the intercostal spaces become wider, the edges of the ribs become

less clearly defined and the skin may be oedematous even in acute hepatitis without suppuration. At an early stage the breath sounds become feeble and the percussion note impaired at the right base. The x-rays show that the right side of the diaphragm is abnormally high, and when suppuration occurs its movements are diminished and finally cease completely. If the diaphragm is perforated by an abscess of the liver a pulmonary abscess or less frequently an empyema develops. In the former case large quantities of thick reddish pus are expectorated, and, if emetine is given, rapid recovery generally results.

An abscess in the anterior and lower part of the right lobe produces a tender tumour in the right side of the epigastrium, the lower ribs become prominent, and the lower border of the liver can either be felt or is found by percussion to be displaced downwards. A rub may be heard when an abscess reaches the surface of the liver but it disappears on the formation of adhesions with the anterior abdominal wall. In advanced cases the skin becomes oedematous, fluctuation is present, and the abscess may open externally just below the costal margin. Less frequently the abscess reaches the under surface of the liver when it may rupture into the duodenum or hepatic flexure pus is then passed by rectum, and the tumour suddenly diminishes in size. The general condition of the patient immediately improves the temperature falls and spontaneous recovery may follow. In a case I saw in Salonica the rupture of a hitherto unsuspected abscess into the lung and intestine at the same time led to rapid recovery.

An abscess in the left lobe of the liver generally gives rise to a tender tumour in the epigastrium. An abscess bulging from the lower or upper surface of the left lobe is less easily diagnosed, it may escape recognition till it ruptures respectively into the stomach when the characteristic thick reddish pus is vomited, or into the left lung or pericardium.

In some cases the x rays show a localised increased density in the hepatic shadow especially if the abscess is in the left lobe or if it is large and surrounded by a thick fibrous capsule. Localised subdiaphragmatic, subhepatic and retroperitoneal abscesses are uncommon complications, and general peritonitis rarely develops.

#### Diagnosis

The possibility of amoebic hepatitis or hepatic abscess should be considered whenever an individual, who is living or has lived

in a country where amœbic dysentery is endemic, is suffering from progressive deterioration in health with more or less pyrexia, especially if the latter is remittent and accompanied by chills and sweats, and if obscure abdominal symptoms are present

Subacute hepatitis is most commonly confused with malaria, but the rise of temperature in hepatitis is generally in the evening instead of during the day, and the liver is enlarged out of proportion to the spleen instead of *vice versa*, leucocytosis is present with a relative increase of polymorphonuclear cells and only 2 to 4 per cent large mononuclear cells in contrast with the normal or sub-normal count with 15 to 20 per cent. large mononuclear cells found in malaria, the malarial parasite cannot be found in the blood, and the injection of emetine is followed by improvement, whilst quinine fails to influence the pyrexia

A localised tumour in the liver in a man who has been exposed to amœbic infection is generally an abscess and more probably amœbic than a suppurating hydatid cyst. It is elastic or fluctuating unlike the hard solid tumour formed by a gumma and a growth

### Prognosis.

Until 1907 hepatic abscess was the second commonest cause of death in the British Army in India, but the incidence of the disease has become much smaller since amœbic dysentery has been treated by emetine injections, and the mortality has been greatly reduced since the necessity for operation has become comparatively rare owing to the frequency with which treatment is instituted in the pre-suppurative stage. The prognosis is best if the abscess is single, as multiple abscesses are difficult to locate, and their evacuation may be impossible, especially in the rare fulminating cases

### Treatment.

In acute hepatitis without suppuration very rapid improvement follows the subcutaneous injection of 1 grain emetine hydrochloride on twelve or more consecutive days; the hepatic tenderness diminishes within 8 hours of the first injection, and the temperature may fall to normal in 24 hours. As there are no definite signs which distinguish acute hepatitis without suppuration from hepatic abscess unless a definite tumour is present, no local treatment should be adopted until emetine injections have been given daily for a week without producing any improvement. It seems probable that a small abscess may be completely absorbed without aspiration after

the amœbæ in its walls have been killed by subcutaneous injections of emetine (Rogers, 1913)

The most satisfactory treatment of an abscess is evacuation by aspiration, emetine being injected subcutaneously in order to kill the amœbæ in the abscess wall and in any ulcers which are still present in the colon. If the pus reaccumulates, the same treatment should be repeated. The results with this treatment are so successful that the open operation, which is frequently followed by prolonged convalescence or death owing to secondary infection, is required only in the comparatively rare cases in which the situation of the abscess cannot be determined. It is interesting however to note that Larrey Napoleon's surgeon opened hepatic abscesses in over a dozen French soldiers in the Egyptian campaign of 1800 without a death. When an abscess has been opened or has ruptured into the lung stomach or bowel subcutaneous emetine injections should be continued until the temperature has remained normal for a week

### Chronic Amœbic Hepatitis

Amœbic dysentery is almost always associated with hepatic insufficiency as demonstrated by the levulose test although in a large majority of cases there is no sign of hepatic disorder. As acute and subacute hepatitis and hepatic abscess are common complications of amœbic dysentery, it seems reasonable to assume that the functional deficiency of the liver in these cases is caused by a mild and latent form of amœbic hepatitis.

#### Symptoms

It is not uncommon to find hepatic insufficiency in patients complaining of unfitness since returning from a country in which amœbic dysentery is endemic. Some have had definite dysentery but others have had nothing more than a few attacks of diarrhoea, and occasionally they have never had intestinal symptoms of any kind. Constipation is more often present than diarrhoea but slight attacks of diarrhoea lasting for a few hours may follow indiscretions in diet or indulgence in alcohol.

The patient complains of general unfitness and is easily fatigued. Very often he rightly describes himself as liverish. He generally suffers from a constant ache over the liver. Headache is common, and he may complain of aching limbs and lumbago. His appetite is poor and he is below weight. He is pale and sallow though

generally not anæmic, and true jaundice is never present. There is no pyrexia, and the leucocyte count is normal. The whole of the exposed surface of the liver is tender, and the lower border is often more easily palpable than normal, but there is very little enlargement. Amœbic cysts are very rarely found in the stools, but in a small proportion of cases, in spite of the absence of intestinal symptoms, the sigmoidoscope has revealed the presence of small but typical amœbic ulcers—raised red spots with a necrotic centre on a normal pink mucous membrane, very closely resembling boils on the skin. Not infrequently the cæcum and ascending colon are tender, and the stools may contain traces of occult blood, presumably the result of chronic amœbic typhlitis.

### Diagnosis and Treatment.

The diagnosis is quickly confirmed by the result of treatment. The injection of 1 grain of emetine hydrochloride every night for twelve nights results in the disappearance of all symptoms, and the lævulose test shows that there is no longer any hepatic insufficiency. In one case, for example, the rise in the blood sugar was 55 mgm per 100 c c one hour after taking 50 gr of lævulose and in two hours it had only fallen 25 mgm, after treatment the one-hour rise was 6 mgm per 100 c c, and in two hours the blood sugar was the same as before the lævulose had been taken. Further courses of six or more injections should be given 3, 6 and 12 months later in order to prevent recurrence.

### LAMBLIA DIARRHŒA

*Lamblia* (or *Giardia*) *intestinalis* is a flagellate protozoon, which inhabits the duodenum and jejunum and occasionally gains access to the gall-bladder by way of the bile ducts. It is pear-shaped, 10–18  $\mu$  long, with eight flagella, two nuclei, and a sucking disc, with which it attaches itself to the intestinal mucous membrane (Fig 31, 4a). The glands of the small intestine of animals have been found packed with lamblia. There is no doubt that it may give rise to catarrhal enteritis, but it never causes ulceration.

The active lamblia may become encysted in the small intestine, and the cysts, which contain two or four nuclei, are passed in the fæces (Fig 31, 4b). Infection occurs, as in amœbic dysentery, by contamination of food or drink with cyst-containing fæces.

The distribution of lamblia is world-wide, but it is much more common in the tropics and subtropics than in temperate regions.

*Lambha* cysts are often found in the formed stools of healthy individuals, especially in hot climates but the incidence of the infection is much greater in patients suffering from diarrhoea or dysentery. In the last war *lambha* was found in the stools of many British soldiers suffering from diarrhoea or dysentery in Egypt, Gallipoli, Lemnos and Salonica. But in almost every case of dysentery in which it was discovered the *E. histolytica* or *Bact. dysenteriae* was also found if the stools were examined during the early stages of the illness. This, however is not the case with simple diarrhoea, in which *lambha* is often found without any other pathogenic organisms.

*Lambha* can give rise to recurrent attacks of diarrhoea with abdominal distension and discomfort, which may be accompanied by the passage of large quantities of yellowish mucus containing enormous numbers of the active flagellate. In such cases the diarrhoea is almost certainly the result of *lambha* enteritis. I have also seen a good many cases in England, in which mild attacks of abdominal discomfort with slight diarrhoea without the passage of mucus were associated with the presence of *lambha* in the stools. It seems likely too that the diarrhoea, which may persist in cases of dysentery after the primary infection has disappeared is the result of simultaneous infection with *lambha* when this organism is found to be still present in the stools.

*Lambha* infection of the gall bladder may give rise to symptoms of cholecystitis. I have seen one such case in a patient who had never been to any tropical or subtropical country. In this case, as in several described by Lyon, the bile obtained through a duodenal tube after injecting magnesium sulphate contained enormous numbers of very active *lambha* (Fig 31, 5). At least four cases have been recorded in which the bile obtained from a chronically inflamed gall bladder immediately after operation contained numerous *lambha* (*vide* de Muro 1939) and one similar finding has been made *post mortem* (Caldor and Rigdon, 1935).

#### Treatment

Of the many drugs which have been tried in the treatment of *lambha* atabrin, as first recommended by Galli Valerio and de Muro is much the most effective. One 0.1 gm. tablet is given three times a day after meals for five days. This always results in the disappearance of the organism both from the duodenal contents and the stools, and the disappearance is generally permanent.

## POST-DYSENTERIC COLONIC IRRITABILITY

Patients who have recovered from an acute attack of dysentery frequently remain unfit for a considerable period, and irritability of the bowel may remain for years. The symptoms are at first caused by a non-specific chronic colitis, which may follow either bacillary or amœbic dysentery after the specific infection has died out, but the possibility that dysentery bacilli or amœbic cysts may still be present can only be excluded by frequent expert examinations of the stools. In the later stages sigmoidoscopy and examination of the stools show that there is no actual colitis present, the condition being due to a functional irritability of the colon. The patient suffers from alternating attacks of constipation and diarrhoea, the latter often brought on by aperients taken for the relief of the former, or it may follow an indiscretion in diet or exposure to cold. The diarrhoea is accompanied by colic, which may be severe. The fluid fæces contain much undigested food, often with mucus containing columnar epithelial cells, but pus cells and blood are present only in cases in which there is still active colitis. The diarrhoea may last for a few hours only, or it may continue for two or three days, the attacks being separated by intervals of several weeks or months. Sometimes slight diarrhoea is continuous.

In the early stages the patient has little appetite and cannot regain his former weight. He complains of constant abdominal discomfort. Slight tenderness is often present, especially over the iliac colon, which can generally be felt as a firmly contracted cord. There is no fever, but the pulse is often rapid, and symptoms of "effort syndrome" may be present. The patient gets quickly tired and may complain of headache. All the symptoms are aggravated by physical activity.

After a time the general symptoms disappear, but a tendency to diarrhoea with slight dietetic indiscretions, exposure to cold and mild general infections may remain for many years, and I still frequently see patients whose bowel irritability dates from an attack of bacillary dysentery contracted in the last war.

"Gippy tummy" is the popular name given in Egypt to a very common disorder characterised by short attacks of colicky abdominal pain accompanied by explosive diarrhoea. It is frequently brought on by nocturnal abdominal chill, the patient having worn

too little covering for the abdomen during the cool nights which frequently follow hot days. But attacks probably never occur in the absence of latent infection with either *Bact. dysenteriae* or *E. amarae*.

### Prevention and treatment

Except in mild cases of dysentery of very short duration, it is advisable for the patient to live for some months after his recovery in a country with a warm and equable but not too hot climate. If this is impossible it is of great importance to guard against chills when the sun goes down and a rapid fall in temperature occurs, and the patient should wear a cholera belt from sundown till morning. He should take no green vegetables except as purées, no salads nor pickles, and no fruit skins nor pips, whether raw cooked or in jam, for at least six months after the symptoms have disappeared. Smoking is permissible only in strict moderation. He should chew very thoroughly and any pyorrhoea should be thoroughly treated in order to prevent secondary infection of the bowel.

The bowels should be kept regular by an unirritating vegetable mucilage such as isogel (one teaspoonful morning or evening or both) or by liquid paraffin. The dose should be just sufficient to produce a single stool or at most two stools. All other aperients must be avoided.

Some of the post-dysenteric symptoms may be due to achlorhydric gastritis. A test-meal should therefore be given, and if achlorhydria and excess of mucus are found, treatment for two or three weeks with an ulcer type of diet and lavage each morning with hydrogen peroxide should be instituted, as in 80 per cent of cases this results in a return of normal secretion, which is often followed by disappearance of the diarrhoea. If the achlorhydria persists, the patient should take a drachm of dilute hydrochloric acid (B.P.) in about five ounces of water to which the juice of an orange with sugar or some other flavouring agent is added, before breakfast and as a beverage with the midday and evening meals.

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## CHAPTER XXII

### EPIDEMIC JAUNDICE

Epidemics of jaundice have occurred in every war. Larrey gives a good description of a severe epidemic among the wounded in Cairo during Napoleon's Egyptian Campaign in 1800. Among the 2,218,559 men in the Federal Army during the American Civil War there were 22,509 cases of jaundice with 161 deaths, and 799 cases occurred amongst 33,380 Bavarian troops stationed near Paris between February and May 1870. In the South African War there were 5,648 cases with a very small but unknown mortality. In the war of 1914-18 epidemics of varying size and severity occurred on every front and in the present war it has been more prevalent than ever before.

There are two and perhaps three entirely different diseases which may give rise to epidemics of jaundice. These are infective hepatitis, leptospiral hepatitis, and perhaps true catarrhal jaundice. It is difficult in retrospect to decide which variety was prevalent in the earlier campaigns. There is however no doubt that leptospiral jaundice accounted for a large majority of the cases in the British, French and German armies in France and Flanders from the spring of 1916 to the end of the last war but it did not occur anywhere else and has not reappeared in the present war. There is equally no doubt that all the epidemics of the present war have been caused by infective hepatitis, which is probably produced by a virus and has no connection with the spirochaetal disease. Infective hepatitis also occurred in the German army in the last war. There is some evidence, which however is not conclusive, to show that the epidemic of jaundice in Gallipoli between August and December 1915 was of the catarrhal type but it is uncertain whether the epidemic which occurred in Mesopotamia during the hot weather of 1916 and 1917 was the same disease.

#### I INFECTIVE HEPATITIS

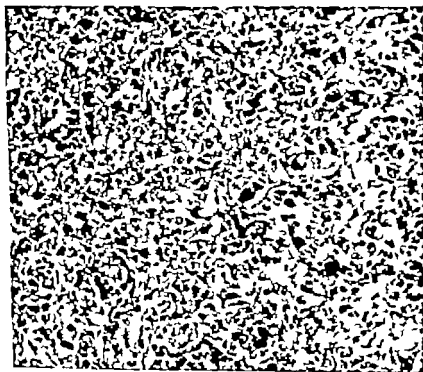
By SIR ARTHUR HURST AND COL. H. B. F. DIXON

Infective hepatitis has become increasingly common during the last fifteen years both in Great Britain and abroad (Cullinan 1939). It is now the most important infection in the British army in

North Africa, Malta, Syria, and Palestine The high incidence among British troops in Malta (11·5 per thousand in 1940) is in striking contrast with the low incidence among Maltese (0·7 per thousand) (Dixon), and among British and New Zealand troops in North Africa compared with Indians and Maoris (Illingworth, 1943) There was an enormous incidence in a certain five-mile stretch of the line in North Africa, whilst New Zealanders were in occupation, but none in the neighbourhood They were replaced by Indians who did not get it, and the Indians in turn by British, in whom it became very common again after about a month (Illingworth, 1943) Infective hepatitis is also common in the United States and in Iceland, and widespread epidemics have occurred in the German army on all fronts and in all the occupied countries (Gutzeit, 1942) The disease should be called infective hepatitis rather than epidemic or catarrhal jaundice, as it is certainly infective in origin and actual inflammation of the liver is always present The old name of catarrhal jaundice should be used only when the jaundice is the result of obstruction of the mouth of the common bile duct secondary to duodenal catarrh and the liver is not primarily involved Infective hepatitis is a better name than infective jaundice, as many cases occur during epidemics which are so mild that no jaundice develops (van Rooyen and Gordon, 1942)

### Pathogenesis

The specific infective agent of infective hepatitis has not been discovered, but it is believed to be a virus, which is disseminated by droplet infection, often by healthy carriers and patients with sub-clinical attacks It is often possible to trace contacts Thus husband and wife, parents and children, and men in the same billet or platoon may be infected The long incubation period, the immunity after an attack, the occurrence of sub-clinical attacks which confer immunity, and the absence of leucocytosis all point to a virus infection In the hepatitis following serum injections the inflammatory material can pass through bacteria-trapping filters and withstand the action of phenol and freezing, though it is destroyed by heat, and it can be propagated in serum-tyrode-chicken-embryo-medium Thus it behaves as a virus, though its further investigation is handicapped by the failure to transmit it to any animal

Fig 32.—Infective hepatitis. ( $\times 100$ .)

One month later, it was very little reduction of the bipolar pattern in discharges of the 1st sacral segment in the same nerve—evidently remains in the partial state, H & L.  
(Duke W. School and Hospital)

[illegible]



FIG 33—Red blood cell distended by a trophozoite of *P. vivax* which has reached the middle stage of its development ( $\times 1000$ )



FIG 34—Rupture of a red cell with liberation of fully developed merozoites, free to enter fresh red cells and restart the cycle ( $\times 1000$ )

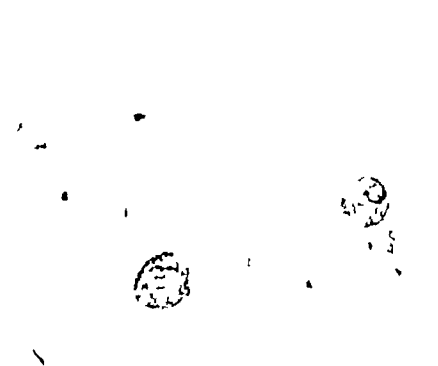


FIG 35—Blood film of benign tertian malaria (*P. vivax*), taken during early sweating stage ( $\times 850$ )

Two young trophozoites, one in ring stage the other further developed. The red cells are pale and show anisocytosis and polychromasia

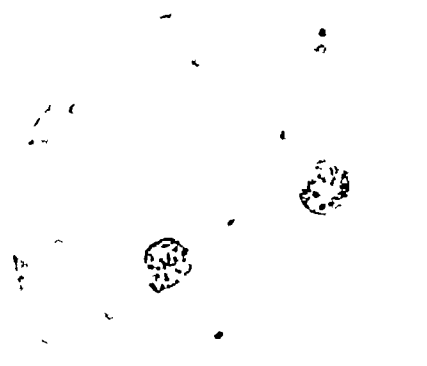


FIG 36—Blood film in benign tertian malaria (*P. vivax*), taken during the fever free period ( $\times 850$ )

The trophozoites have grown until they almost fill the red cell and early differentiation of the merozoites is now visible



FIG 37—Trophozoites of *P. falciparum* (fatal malignant tertian malaria), in early ring form ( $\times 850$ )

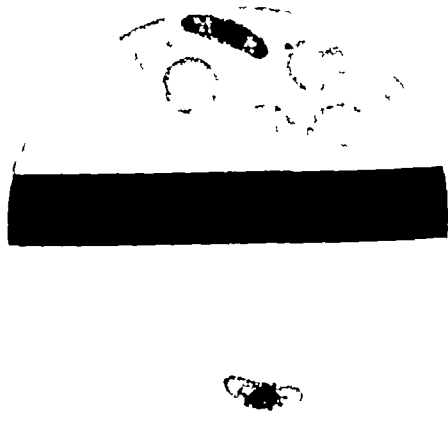


FIG 38—Gametocytes (crescents) of *P. falciparum* the male form above, the female below ( $\times$ )

There is no doubt that alcohol lowers the resistance of the liver to infection with the virus of infective hepatitis. This probably explains the much higher incidence in officers than in other ranks and in men than in women. In Malta in 1940 it was 40 per 1 000 in officers compared with 11.5 in men, and out of 600 cases seen not more than 20 or 30 were in women (Dixon) although the incidence among children in England is the same in both sexes.

The disease is probably infectious only during the few days before the jaundice appears. Consequently there is never any cross-infection in medical wards to which cases of jaundice are admitted, though it is frequent in surgical wards when patients are admitted in the pre-icteric stage for supposed surgical conditions such as appendicitis or pyelitis, and in wards where cases are admitted for possible sand fly fever before the appearance of jaundice (Dixon). The incubation period is usually three to five weeks with a minimum of three weeks. In the infective hepatitis following treatment with arsaphenamine and inoculation of vaccine containing human serum the incubation period is longer generally between six and eighteen weeks.

### Morbid Anatomy

Post-mortem examination in the numerous fatal cases which occurred in the very extensive epidemic of jaundice in Scandinavia in 1926 and 1927 always showed acute or subacute hepatic necrosis. Similar changes were found in the very small number of fatal cases occurring in the slight epidemics of mild infective jaundice in Great Britain since 1918 and Colonel Sidney Smith (1942) found advanced hepatic necrosis in eight fatal cases in British soldiers in the Middle East in 1942. Eppinger had found subacute hepatic necrosis with no catarrh of the bile ducts in three jaundiced Austrian soldiers who had died of tetanus during a mild epidemic of jaundice in 1916.

Roholm and Iversen (1939) have shown by means of aspiration biopsy that infective hepatic jaundice is a true hepatitis and not a non-inflammatory hepatic necrosis, as had been generally assumed from the results of post-mortem observations. They examined thirty-eight specimens obtained during life from twenty-six sporadic cases occurring in Copenhagen. Dible, McMichael and Sherlock (1943) using exactly the same technique obtained similar results in all of a series of cases examined in London. Inflammatory changes were invariably present in the connective tissue and the

parenchyma showed irregular foci of necrosis. The trabecular structure of the liver was destroyed, but the interlobular bile ducts and Kupffer cells were unaffected. The inflammation generally subsided within a month of the onset of jaundice. In mild cases all signs of disease eventually disappeared. In other cases a varying degree of hyperplasia of connective tissue persisted. In one case, in which death occurred in coma on the fifteenth day, specimens obtained by the same method sixteen hours before and ten hours after death were compared by Roholm and Iversen, the latter showed advanced necrosis of the parenchymatous cells which failed to stain, in striking contrast with the former in which the cells stained well. They concluded that the severe necrosis found after death in fatal cases is to a large extent caused by post-mortem autolysis. The jaundice is presumably in part a result of impaired functional activity of the liver cells, which normally take up the bile pigment from the Kupffer cells and excrete them into the bile capillaries, and in part a result of disorganisation of the structure of the lobules with consequent rupture of the intralobular bile capillaries, which are lined by the hepatic cells and have no independent epithelial walls.

### Symptoms.

In very mild cases jaundice is the only symptom. The illness generally begins with headache, pains in the limbs, anorexia and malaise with fever. The initial symptoms closely resemble those of sand-fly fever, but the headache is less severe and less frequently localised in the eyes. Anorexia is always present and is often profound, it is generally accompanied by nausea but vomiting is uncommon. This may give place to excessive appetite about ten days after the onset, Illingworth (1943) noted that almost all of three hundred patients seen in North Africa wanted enormous quantities of food—far more than anybody else in the wards. The patient has a nasty taste in his mouth and has no desire to smoke. There is no herpes labialis. Abdominal discomfort, especially in the epigastrium and hepatic region, is common. The temperature is generally raised for three to six days, but mild cases with no symptoms except jaundice are generally afebrile.

One to eight days after the onset jaundice develops and the pre-icteric symptoms and fever rapidly disappear. The stools are always pale and the urine contains bile, which can generally be found one or two days before the appearance of jaundice. Men

reporting sick with abdominal symptoms during an epidemic of jaundice should therefore have their urine examined for bile. The jaundice persists for five to seventy days with an average of three weeks. For two or three days before its appearance and in cases in which there is no clinical jaundice throughout the illness the wheal produced by the intradermal injection of 0.25 c.cm. of 1 per cent histamine is discoloured yellow (Klein, 1931, Cullinan and Brodribb 1936). The rapid pulse of the pyrexial period is replaced by bradycardia and depression is common so long as the jaundice lasts, but pruritus is rare.

The liver is generally tender and harder than normal. It is enlarged in about 60 per cent of cases. It often remains enlarged after the urine is bile-free and the jaundice has disappeared. The icteric index remains high for some time, generally about a month, after apparent recovery and disappearance of bile from the urine, indicating that the liver is still abnormal (Illingworth, 1943). This may be associated with considerable enlargement of the liver with final recovery in most cases in three to five months. The spleen is enlarged and harder than normal in about 30 per cent of cases.

Leucopenia with relative lymphocytosis is common, and there is never leucocytosis. Slight anaemia may develop. Haemorrhages do not occur.

Relapses are rare. They are more frequent in England than overseas. They are generally more severe than the original attacks and are almost invariably the immediate sequel of indulgence in alcohol. Second attacks are rare.

No deaths occurred in Palestine or Malta and very few in North Africa and England.

#### JAUNDICE FOLLOWING ARSAPHENAMINE TREATMENT OF SYPHILIS

Concurrently with the world wide increase in the incidence of infective hepatitis since 1939 there has been a striking rise in the number of patients developing jaundice during treatment for syphilis. In a series of 1 965 soldiers treated for syphilis between 1940 and 1943 297 or 15 per cent developed jaundice (Campbell, 1943). In 46 per cent the jaundice occurred during or immediately after the first course of treatment, in 37 per cent during the second course and in the remainder during the third or fourth course. The clinical and pathological features of cases of this kind, admitted into the same wards at the Connaught Hospital as those with

infective hepatitis, were indistinguishable from the afebrile form of the latter (Dixon), and Dible, McMichael and Sherlock (1943) found that the pathological appearance of the liver in biopsy specimens was identical. The fact that less than 3 per cent of the patients with jaundice also developed dermatitis and none albuminuria shows that arsenical poisoning is not the sole cause of the hepatitis. The recent rise in the incidence of jaundice in patients undergoing anti-syphilitic treatment is probably due to the increased liability to infective hepatitis, which results from the toxic action of syphilis on the liver and from treatment with arsenic, which is also a liver poison. The vulnerability of the liver is perhaps further increased by some vitamin deficiency due to war-time lack of cheese, milk and butter, and in some cases by the toxic effect of alcohol.

The prognosis is good, but it is unwise to give further arsenic injections for at least three to six months however completely the patient may appear to have recovered from the hepatitis. Each case should then be judged on its merits, depending on the severity of the hepatitis and the dose of arsenic already given. Of course bismuth injections can be continued throughout.

#### SERUM JAUNDICE

In 1936 96 cases of jaundice with one death occurred after immunisation against yellow fever. Since the introduction of a new vaccine in 1937 there were no further cases (Findlay and MacCallum, 1937). A very serious outbreak followed the use of yellow fever vaccine made with human serum in Brazil in 1939 and in the United States in 1942 (Fox *et al*, 1942). In Brazil there were 1,072 cases with 24 deaths, in the States 28,585 cases with 62 deaths. The vaccine was essentially the same as that used for several million inoculations since November 1937. The clinical features of the illness closely resembled those of the afebrile form of infective hepatitis, except that the incubation period was prolonged to six to eighteen weeks. The temperature was usually normal or only slightly raised and there was no leucocytosis. Joint pains, urticaria, and enlargement and tenderness of the liver each occurred in 20 per cent of the American cases. Only 0.2 per cent ended fatally, generally two to six weeks after the onset. Recovery was generally complete in four to eight weeks, and permanent liver damage was rare. In fatal cases lesions similar to those found in infective hepatitis and arsaphenamine jaundice were discovered,

together with marked cedema of the gastro-intestinal tract enlargement of the spleen and hæmorrhages in serous membranes.

All attempts to transmit the disease into animals having failed, as with ordinary infective hepatitis, Findlay and Martin instilled nasal washings from their patients in the pre-icteric or early icteric stages of jaundice following yellow fever immunisation into the noses of three volunteers. Jaundice with symptoms similar to those of infective hepatitis, followed after incubation periods of twenty-eight thirty and fifty days.

A similar epidemic occurred in England in 1937 in several scattered groups of children after inoculation with measles, convalescent serum coming from a single presumably contaminated source (McNalty 1938). Jaundice developed in 37 out of about 100 receiving the serum and seven died.

#### Treatment.

Experimental observations on animals with infective hepatitis and clinical observations on the disease in man indicate that the most suitable diet is one containing abundant carbohydrates to maintain the glycogen deposits in the liver cells, abundant proteins to compensate for the low plasma protein observed in the disease and a minimum of fat, together with abundant fluids. Abundant carbohydrate can be supplied in the form of glucose, half a pound of which in a pint of water, preferably flavoured with fruit juice should be given *per diem*. In the rare cases in which vomiting occurs or the patient is too drowsy to take sufficient fluid, normal saline alternating with 10 per cent dextrose solution should be given intravenously by the drip method. Not more than 500 c.cm. of the latter should be used, and 10 units of insulin should be injected subcutaneously at the same time.

The patient should not be allowed up until bile is no longer present in the urine, the jaundice has completely disappeared, and the liver and spleen are no longer palpable. After recovery the patient should be advised to remain teetotal for at least a year and thereafter to be very moderate, always avoiding strong alcohol on an empty stomach, as the liver is likely to be permanently more vulnerable than normal and consequently more liable to become cirrhotic with a comparatively moderate consumption of alcohol.

## II LEPTOSPIRAL HEPATITIS

Leptospiral hepatitis, commonly called spirochætal jaundice, occurred in a mildly epidemic form among British French and

German troops in France and Flanders from the spring of 1916 to the end of 1918. So far it has not reappeared in the present war. It is sometimes known as Weil's disease, but there is no evidence that the four sporadic cases of fever with jaundice, enlargement of the liver and spleen, and nephritis, described by Weil of Heidelberg in 1886 was spirochætal in origin, and epidemics of similar cases had already been described by several French physicians since Larrey's account of the Egyptian epidemic of 1800. The disease has long been common in Japan. In recent years it has been recognised among fish-workers, especially in the north-east of Scotland (Davidson *et al*, 1939), and coal-miners and sewage workers in different parts of Great Britain.

### Ætiology and Pathology.

In November 1914 Inado, Ido and their fellow-workers showed that the epidemic jaundice which every year attacked between 3,000 and 4,000 miners in Japan was due to infection with a spirochæte, now known as *Leptospira ictero-hæmorrhagica*, which is present in the blood during the first week and the urine during the first four weeks. Animals develop jaundice on inoculation with the blood of a patient, and the organism can be recovered from their blood and viscera. Very soon after the results of the Japanese observations became known in France, a number of investigators succeeded in isolating the organism from the blood and urine of soldiers suffering from infective jaundice, and they confirmed the animal experiments.

Noguchi (1917) later found that the strains of *Leptospira ictero-hæmorrhagica* isolated from patients in Japan and Belgium and from rats in America were identical in their morphological and serological properties. He showed that it differs from all other spirochætes in possessing a large number of minute spirals, 10 to 12 to each  $5\mu$ . But the characteristic features under the microscope are its tapering ends and tendency to be bent at an angle, thus producing forms like the letters S, C or L. This spirochæte is actively mobile and can be cultivated easily at about  $30^{\circ}\text{C}$  in the surface layers of semi-solid blood-agar.

The morbid anatomy of the disease was studied in four fatal cases by Stokes, Ryle and Tytler (1916), in three by Dawson and Hume (1916), and in five by McNee (1920). Varying degrees of hepatic necrosis together with round-celled infiltration of the interstitial tissue was present. The appearances were essentially the same as in infective hepatitis except for the presence of leptospiræ.

The kidneys may show nothing more than cloudy swelling whilst in other cases there is inter and intra tubular infiltration with polymorphonuclear leucocytes, sometimes with hæmorrhages in the glomeruli and degeneration of the tubular cells (Dawson (1918)). The spirochæte is found in the liver kidneys and suprarenal glands after death.

The leptospira was found in the kidneys and urine of wild rats in the infected areas of Japan, and Stokes discovered it in rats caught in the trenches in France in which the disease had occurred. It is found in about 25 per cent. of rats in Great Britain, both in places where the disease is unknown and in those, such as the north east of Scotland where it is comparatively common. It has also been found in rats in districts in France where the disease is unknown. The infection in rats is chronic, the leptospira being excreted in the urine for long periods. The infected urine of rats is the main source of the *Leptospira ictero-hæmorrhagica* in man, and the urine of patients suffering from the disease is a secondary source.\*

Infection can be produced experimentally through the alimentary canal, and through abrasions on the surface of the body and even apparently healthy skin. As the organism can live for a time in water the infection probably occurs under natural conditions through both of these channels. When several cases develop within two or three days among men living together in whom infection by the skin can be excluded, the infection must have occurred by mouth. Cases have been observed in Japan, in which the disease began with tenderness and enlargement of the femoral glands, about a week after the corresponding foot had been wounded whilst the man was working barefoot or in sandals in stagnant water or wet earth. The incidence of the disease greatly diminished in Japan after the mines had been thoroughly drained. It occurred in France only among troops who occupied certain badly drained trenches, especially during wet weather. When a unit was moved to a different part of the line no further cases occurred, but the disease quickly appeared in the unit which replaced it in the infected trenches.

There is no evidence that mosquitoes, fleas or lice are in any way concerned in the conveyance of the disease, and no cases have

\* A serious outbreak of leptospiral hepatitis occurred in Samoa in 1939-40. This was almost certainly the result of contact with pigs, in which the disease is endemic in Samoa. The possibility that farm animals as well as rats may be carriers in other countries calls for investigation. (D W Johnson (1943) *Brit. med. J.*, 2 669)

been observed in which the infection appeared to pass direct from man to man

### Symptoms.

The incubation period is short. A colleague of Martin and Pettit (1919), who was engaged in transmitting the infection from one guinea-pig to another on September 2nd and 4th, was seized with fever on the 10th and became jaundiced on the 16th, the leptospira being isolated from his urine on the 17th.

The onset is most frequently gradual with a feeling of general unfitness, headache, pains in the back and limbs, unsteadiness or inability to stand or walk, anorexia, nausea and often vomiting. Sometimes, however, it is sudden with shivering, faintness or giddiness, and great prostration. The temperature rises to between  $102^{\circ}$  and  $105^{\circ}$ , it varies irregularly between  $100^{\circ}$  and  $103^{\circ}$  for a week, and then descends by lysis, becoming normal between the tenth and fourteenth day. A slight return of fever often occurs a few days later, it may last for ten to fifteen days, and is generally unaccompanied by any exacerbation of symptoms.

Jaundice appears between the second and seventh day and increases up to the ninth or tenth day, when it generally fades rapidly, though occasionally it is more persistent. The skin, especially of the face, has frequently a characteristic red-yellow colour owing to simultaneous capillary dilatation. Pruritus is absent or slight. The degree of jaundice does not vary with the intensity of the toxæmia. In several cases symptoms exactly similar to those of leptospiral hepatitis, except for the complete absence of jaundice, have been observed in patients whose blood and urine contained the leptospira and whose blood produced leptospiral hepatitis when injected into guinea-pigs. Many cases diagnosed as P U O in France were probably of this nature.

The tongue is dry and coated with a brown fur, and sordes are present on the lips and teeth. Anorexia persists until the jaundice begins to disappear. The initial vomiting sometimes continues for several days. In severe cases there may be persistent hiccups. The patient generally complains of vague discomfort or pain over the whole abdomen, which is diffusely tender, especially over the upper half. The liver may be slightly enlarged and tender, but the gall-bladder is not palpable and no special tenderness is present in its neighbourhood. The spleen is generally normal in size, but the lymphatic glands in the axillæ and groins are often slightly enlarged.

The patient complains of lassitude and of pains in the head, eyes, back and limbs. The pain is accompanied by tenderness especially of the calves and back, but every muscle of the body may be involved. In severe cases the myalgia, which is perhaps the most constant symptom of the infection, is extreme—it prevents sleep and the patient feels as if he had been beaten all over and dislikes moving his limbs or turning in bed. The neck may be stiff and Kernig's sign may be present. Costa and Trouner found that the cerebro-spinal fluid in cases of this sort was under pressure and contained excess of polymorphonuclear cells and lymphocytes with excess of albumen as well as bile. In three cases the fluid produced jaundice on injection into animals and the leptospira was isolated from their organs. The jaundice was only just perceptible in one of these patients, and herpes was present as well as the other symptoms suggestive of cerebro-spinal meningitis. In fatal cases hepatic insufficiency leads to drowsiness and delirium, followed by twitching convulsions and finally coma. The conjunctivae are pink from capillary dilatation and the chief conjunctival vessels show varicosities. Small subconjunctival hemorrhages may occur. The eyeballs are often tender. Herpes is common on the lips and may spread over the chin and upper part of the neck—it is generally hemorrhagic.

Bile is present in the urine for four or five weeks in the jaundiced cases, and it may also be found in the absence of obvious jaundice. The urine almost invariably contains from a trace to a considerable quantity of albumen, often with granular and hyaline casts and a few red corpuscles. In severe cases the urinary output falls and the blood urea rapidly rises, and the terminal symptoms in fatal cases may sometimes be uraemic and not hepatic in origin. When jaundice is absent, the case may be mistaken for war nephritis, but oedema and the characteristic dyspnoea are not present. Retention of urine is occasionally observed. Extreme constipation is common, and the faeces are sometimes clay-coloured, but more frequently contain sufficient bile to produce a light-brown colour.

The heart is not enlarged, and the pulse is generally slow in proportion to the temperature, even when there is no jaundice. Slight bronchitis is sometimes present, especially in severe cases when the sputum is generally either rusty or streaked with bright-red blood, but broncho-pneumonia and lobar pneumonia have not been observed. In addition to haemoptysis a little blood may be

present in the vomit and rarely in the fæces. Epistaxis is an occasional symptom, and purpura may occur in severe cases. In mild epidemics hæmorrhages are so rare that the name *Spirochaetosis ictero-hæmorrhagica* is inappropriate. Among Moreschi's 361 cases with a mortality of only 0.55 per cent. no hæmorrhages were observed apart from epistaxis.

The number of red corpuscles and percentage of hæmoglobin are often reduced, especially when there is severe toxæmia. Slight polymorphonuclear leucocytosis is constantly present, the total number of leucocytes being between 10,000 and 30,000 and the percentage of polymorphonuclear cells about 85 per cent.

### Pathological Diagnosis.

Between the fourth and ninth day the spirochæte may be found in the blood, but it is present in such small numbers that this method of diagnosis generally fails. The same drawback is present with regard to the urine, in which the organism may sometimes be found after the second week if searched for at intervals of a few days. It is important to remember, however, that non-pathogenic spirochætes are not uncommon in the urine, thus Stoddard isolated spirochætes from the urethra in 56 per cent. of 50 soldiers suffering from various medical and surgical diseases and 22 per cent. of 50 healthy soldiers. As the incubation period in guinea-pigs is six or more days, the diagnosis cannot be confirmed by animal experiments in less than eight days. The peritoneal cavity of a guinea-pig should be inoculated with 3 to 5 c cm of the patient's blood in the early stages of the disease. From the seventh to the twenty-eighth day the centrifugalised deposit from 50 to 250 c cm of recently passed urine suspended in 5 c cm of normal saline solution may be used instead, but it gives a positive result in a smaller proportion of cases. The animal develops fever, jaundice and hæmorrhages after six to twelve days. Death always follows.

Martin and Pettit (1919) showed that a diagnosis can be made serologically, as a specific agglutinin develops in the serum, which is still present for at least 22 months. Dilutions of the patient's serum and a suspension of the leptospira are mixed in small tubes at 32° C for three hours. The mixtures are then examined as thin films under the dark-ground illumination microscope. Agglutination is readily seen in this way together with any lysis of the organisms. The lysis can usually be detected extending to higher dilutions than the agglutination. The recent observations of

Davidson and Smith in Aberdeen have shown that a positive reaction never appears before the third day—it is present in 50 per cent. of cases by the fifth day and in 100 per cent. by the ninth day. The titre of the serum increases from 1 in 10 in the early stage to 1 in 10 000 or even 30 000 by the tenth day.

### Differential Diagnosis

Leptospiiral hepatitis differs from the other forms of epidemic jaundice in the more prolonged pyrexia, the dry furred tongue, the conjunctival congestion, the severity of the muscular pains, the character of the urine, the frequency of herpes and the greater tendency to hæmorrhages. A definite diagnosis of leptospiiral hepatitis can however be made only by discovering the spirochæte in the blood or urine or in the tissues of an inoculated animal.

### Prognosis

In the war of 1914–18 the mortality of leptospiiral hepatitis was between 4 and 5 per cent. in France and Flanders, but was 13 per cent. in Germany and as low as 0·55 per cent. in Italy. The disease in Europe is thus milder than the form occurring in Japan, in which the mortality is about 30 per cent. Dawson and Hume classified 18 of their cases as severe and 58 as mild—in the former the illness lasted about three weeks and in the latter about one, but few patients were fit for duty in less than three months.

### Prophylaxis

Wherever cases of leptospiiral hepatitis have occurred the ground should be disinfected or drained as thoroughly as possible and a special effort should be made to destroy all rats. The urine of patients should be disinfected for nine weeks from the date of onset of the disease.

### Treatment

The same diet as that recommended for infective hepatitis (p. 329) should be given. Epsom salts are required for the constipation and iron for the anaemia. Japanese observers concluded from their animal experiments that salvarsan was of little value, and Dawson and his colleagues found it useless in France.

Dutch investigations have shown that anti leptospiiral serum given within the first four days reduces the severity of the toxæmia and lowers the mortality. It has much less effect after the seventh day when septiciæmia is no longer present. The dose is 10 to 20 c.cm. given intramuscularly or intravenously.

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Prof. Leonard Findlay in Oxford in 1913 developed jaundice after a week of general and digestive symptoms with pyrexia. Biopsy by hepatic puncture showed no abnormality either on the second day of jaundice or ten days later when the jaundice had disappeared. This presumably was a case of true catarrhal jaundice.

### Epidemiology

The first outbreak of jaundice in the last war occurred in July 1915 among the troops stationed in certain camps in Egypt. There was no jaundice at Gallipoli until the middle of August, when it suddenly appeared in a large number of different units in all parts of the Peninsula—it then rapidly spread among the men of each unit. Six weeks later fresh cases appeared in Lemnos and Imbros. This is most easily explained on the assumption that the infection was conveyed to different parts of the Peninsula by convalescent or contact carriers in drafts from the infected Egyptian camps, and that the subsequent spread to the islands was caused by patients coming from Gallipoli. The epidemic reached its greatest intensity in the second half of October—it then gradually declined and finally disappeared at the end of the year. Official statistics give no adequate idea of the prevalence of the disease, as large numbers of men continued at duty throughout their illness, especially in November and December. Several regimental medical officers told me that at times as many as one-tenth of their men actually in the trenches were jaundiced. In one battalion there were a hundred cases during October but only thirty-six were regarded as of sufficient severity to be sent into a field ambulance.

Epidemic jaundice was as frequent among the French as the British at Gallipoli, but no cases occurred among the Turks, although their first line was situated only a few yards from that of the Allies and their sanitary arrangements were much less good.

Epidemic jaundice occurred, though much less frequently among the British and French troops who went from Gallipoli to Serbia in October 1915 and a few cases continued to develop among them after their return to Salonica until the beginning of 1916 when the epidemic ceased. Only a few sporadic cases of catarrhal jaundice occurred among the British troops at Salonica who had come from France or England, and there were no cases among the French who came direct from France. It did not reappear in Egypt after 1915. The disease appeared in Mesopotamia during the very hot weather in June and July 1916 and in 1917—1,538 British and 2,634 Indian

## III. CATARRHAL JAUNDICE

The clinical picture of the epidemic jaundice which occurred in Gallipoli was characteristic of catarrhal jaundice. So far as I know, only three autopsies were performed on Gallipoli cases, as death was very rare. In two intense catarrh of the duodenum and larger bile ducts was discovered (Willcox), and in the third case there was severe entero-colitis and a plug of tenacious mucus was found impacted in the orifice of the biliary papilla (Rolleston).

I obtained the duodenal contents through an Einhorn tube from nine cases of catarrhal jaundice at Lemnos during the Gallipoli epidemic in December 1915, and the material was examined by Colonel C. J. Martin. The fluid was turbid owing to excess of mucus and contained very numerous coliform bacilli, whereas specimens obtained at the same time from normal controls were clear and almost sterile. In 1923 Jones and Minot, in a study of the duodenal contents in twenty-six cases of "catarrhal jaundice," found excess of mucus and large numbers of non-bile-stained cells, chiefly polymorphonuclear leucocytes with some epithelial cells, in the early stages. As the jaundice disappeared and bile pigment reappeared in the duodenum, clumps of bile-stained leucocytes, epithelial cells, precipitated bilirubin and greenish-yellow casts, similar to urinary casts, were found for a short time, but disappeared when the jaundice faded. Jones and Minot suggested that the non-bile-stained pus at the onset indicated an initial duodenal infection, which spread up the biliary tract, as shown by the bile-stained cells and pigment granules in the later stages, reaching as far as the biliary radicles in those cases in which bile-stained casts were found.

Catarrhal jaundice appears to have been at one time the cause of most of the sporadic cases of infective jaundice seen in England (Hurst and Simpson, 1934), but it has become very rare in recent years. In civil life, as in war, the disease is extremely mild, so that it is very rare that an autopsy can be performed. But in 1910 a young woman committed suicide the day after admission in a typical attack of catarrhal jaundice. Eppinger found no abnormality of any kind in her liver, the mouth of the common bile duct was, however, completely obstructed by inflammatory swelling, so that it was impossible to squeeze bile from the distended gall-bladder into the very inflamed duodenum. A child under the care of

anorexia is soon followed and in a few cases is immediately preceded by a feeling of epigastric discomfort which may develop into actual pain, which is constantly present but is increased directly any solid food is taken. It is always accompanied by a moderate degree of tenderness. Contrary to what might be expected the tongue remains clean.

Headache of moderate severity is often present, and may be accompanied by giddiness, which is sometimes so great that the patient is hardly able to stand or walk. In many cases he feels very weak and disinclined to exert himself. These symptoms occasionally precede or develop simultaneously with the gastric symptoms, but more commonly they follow a day or two later.

In the comparatively rare instances, in which the temperature was taken before the onset of jaundice, it was often found to be raised to 100°, and occasionally even to 103° or 104° when the headache was unusually severe with considerable constitutional disturbance.

*Symptoms after the onset of jaundice.*—The patient occasionally notices that his urine is becoming dark, but more often the jaundice is first noticed by his friends, or by the medical officer when he reports sick on account of the premonitory symptoms. The jaundice is never very intense and may be so slight that it is not recognisable except in the conjunctivæ. Its intensity is no indication of the severity of the infection, as pronounced jaundice may occur with no other symptoms, so that one man may be able to continue at duty throughout his illness, whilst another may appear and feel very ill although the jaundice is quite slight. The inflammation of the bile ducts and duodenum does not necessarily produce sufficient obstruction to cause jaundice. Thus Captain Jolly saw several cases at Anzaco, in which the symptoms made him expect that jaundice was about to develop though bile appeared in the urine, no staining of the conjunctivæ or skin occurred. The jaundice reaches its greatest intensity in a few days and then slowly diminishes, a yellow tinge being often visible a fortnight after all other symptoms have disappeared. There is never any complaint of pruritus probably because of the short duration of the jaundice.

The appetite begins to return a few days after the onset of jaundice. Occasionally there is a vague pain in the region of the gall bladder and liver but never in the right shoulder. After a few days the pain disappears, slight epigastric discomfort remain

troops were affected with a mortality of 0.4 per cent. Clinically the Gallipoli and Mesopotamian epidemics were quite distinct from the spirochætal jaundice as it occurred in France and Flanders. Numerous attempts were made to isolate the *Leptospira ictero-hæmorrhagica* both during life and *post mortem* in the Mesopotamian epidemic, but the results were invariably negative. Moreover, there were no rats from which the infection could have been spread.

### Ætiology.

There is some evidence that the disease, like infective hepatitis, is caused by a virus, and it may perhaps be spread by droplet infection. There is no evidence that it is conveyed by contaminated food or water, and dust and flies are not important factors, as the disease was most prevalent in Gallipoli in the autumn and early winter when there were no flies and often no dust.

In view of the extreme frequency of diarrhœa at Gallipoli, it is noteworthy that epidemic jaundice developed most commonly in men whose bowels had been regular for some time before the onset of symptoms, although two-thirds had suffered from diarrhœa, often on several occasions, since their arrival on the Peninsula. Indeed constipation was noted twice as often as diarrhœa as the immediate precursor of the disease.

### Symptoms.

The following description was written in 1916 after seeing large numbers of cases at Lemnos, the majority evacuated from Gallipoli, but some developing on the island. The clinical picture differs in no way from that of the catarrhal jaundice of civil life both in its sporadic and epidemic forms.

In all but about 5 per cent. of cases the jaundice was preceded by other symptoms, the most frequent interval between the onset and the development of jaundice of sufficient intensity to be noticed by the patient or his companions being three days, two days being the next most common interval. In several cases it was between four and seven days, but it was rarely less than two or more than seven.

*Pre-icteric symptoms*—The most common early symptom is anorexia, which may develop gradually in the course of a couple of days, but is often noticed quite suddenly at one meal, generally breakfast, the previous meal having been enjoyed as usual. The anorexia quickly becomes extreme for all solid food, but the patient is generally ready to drink fluids. The mere sight of food makes him feel sick, and if he attempts to eat he is likely to vomit. The

three and six weeks after the onset of symptoms. If his health was already impaired at the time of infection, as was often the case at Gallipoli the added toxæmia is likely to weaken him to such an extent that he will require two or three months to recuperate.

Treatment.

The patient should be kept warm in bed on a fluid diet until his appetite comes back and the abdominal tenderness disappears. He should then rapidly return to a full diet. A dose of Epsom salts should be given at the onset to promote biliary drainage but this should not be repeated unless constipation is present.

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ing for a short time longer Simultaneously the headache and vertigo disappears The bowels tend to be constipated, and considerable relief is experienced when they have been sufficiently but not excessively opened The fæces retain their normal colour in more than half of the cases, in the remainder they become pale and sometimes definitely clay-coloured a day or two after the first appearance of jaundice

Tenderness is almost invariably most marked over the gall-bladder, deep pressure may cause little or no pain with shallow respiration, but on deep inspiration acute pain, which may radiate to the epigastrium, is felt the moment the gall-bladder comes into contact with the fingers The gall-bladder was definitely palpable in about one-tenth of the cases If every patient were examined regularly from the onset of the symptoms, it would probably be felt in a considerably larger proportion of cases, which would be still greater were it not for the tendency of the rectus muscle to contract when palpation is attempted In a few cases the gall-bladder remains enlarged when the tenderness has almost disappeared, it can then be easily palpated

The liver is often slightly enlarged owing to the rigidity of the abdominal muscles this may be recognisable only by percussion, but in about a third of the cases the lower border is palpable, generally about half an inch below the costal margin in the right nipple line The liver is firm and at first moderately tender, but the enlargement may persist after all tenderness has disappeared, occasionally for as long as fourteen or twenty days In a small proportion of cases the spleen is enlarged and slightly tender

As soon as the jaundice appears the pulse becomes slow, it is often only 60, but with the exception of one case in which it fell to 38 the day after the jaundice appeared, rising to 72 four days later, I have not seen a case in which it was below 52 The rate begins to increase again about the fifth day, and it is generally normal in ten to fourteen days

Even if the temperature is high in the pre-icteric period, it falls with the onset of the jaundice, and either becomes normal at once or remains between 99° and 100° for three or four days A higher or more prolonged pyrexia indicates that some other infection is also present

### **Prognosis.**

True catarrhal jaundice is probably never fatal In cases of moderate severity the patient is generally fit for duty between

## CHAPTER XXIII

### MALARIA

BY COLONEL H. B. F. DIXON M.C. M.D. F.R.C.P.

Owing to its extensive geographical distribution and almost epidemic concentration in most warm countries malaria is much the most common of all human diseases. Wherever warmth and water exist together malaria is found, provided there are mosquitoes in which the parasite can develop its full cycle the conditions of temperature and humidity are suitable, and there are people suffering from malaria who can be bitten by the suitable mosquito. The usual limits of the area are between 63° N and 35° S. malignant tertian where isotherm is 70° F., benign tertian where isotherm is 60° F.

In the war of 1914-18 of all diseases responsible for casualties malaria easily took first place. In the British Army alone the admissions to hospital for malaria were almost half a million. In his malarial survey of Greece in 1906 Sir Ronald Ross had said that severe malaria haunts almost every village and the course of almost every stream. It was therefore not surprising that during the war in Macedonia there were 162 000 admissions for malaria with a mortality of 1 per cent. whereas 27 762 was the total for killed, died of wounds, wounded, missing and prisoners. One division alone produced the amazing figure of 5 000 cases of malaria admitted to hospital in one month.

During sixteen months of the campaign in East Africa there were over 100 000 admissions to hospital for malaria, which was responsible for 60 per cent. of the total admissions for sickness during 1917. On one occasion in the Army of the Black Sea during 1918 305 men were sent to man a post near a river within a few weeks 303 of the men were down with malaria and the post had to be abandoned.

In striking contrast with this the health of the British troops in the Middle East was extremely good in 1940 in spite of a trying summer in Africa and Syria. Malaria attacked only 0.58 per thousand compared with 3.5 per thousand in the last war—largely as a result of improved prophylactic measures.

Malaria in war time is no more severe than in peace and

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rose to 13 millions. War may interfere with many civil anti malarial schemes of drainage, which in peace-time keep the malaria rate low with the result that the area concerned may become seriously malarious with many thousands of cases

### The Mosquito

Development of the malarial parasite can take place only in the female of certain species of anopheline mosquito (Fig 41, p 374.) The same species may be a carrier in one part of the world and not in another owing to differences in its habits in different environments.

Of malaria-carrying mosquitoes 95 per cent. never become infected because they do not bite infected persons, they bite persons who are not good infectors or whose blood contains either too few gametocytes or gametocytes all of one sex, temperature and humidity are not suitable for the development of the parasite in the mosquito's stomach and malarial patients in a primary infection are not infectious until the sixth day in benign tertian and the fourteenth day in malignant tertian.

For the development of the malarial parasite in the mosquito a sustained average air temperature of at least 60° F and a relative humidity of at least 63 per cent. are required. The optimum development occurs between temperatures of 60° F and 77° F with a high relative humidity

### The Parasite

Three species of human malarial parasites are well recognised

- (1) *Plasmodium vivax*, producing benign tertian malarial fever known as B T (Figs. 33-36 Plate IV and 39)
- (2) *Plasmodium malariae* producing quartan malarial fever known as quartan. (Fig 40)
- (3) *Plasmodium falciparum* (Figs 37 and 38 Plate IV) producing malignant tertian or subtertian fever known as M T

A fourth malarial parasite, *Plasmodium ovale*, is of relatively small importance.

*Human cycle (schizogony)* --When an infected female anopheline mosquito bites a man the parasites, at this stage of their existence called sporozoites, are injected into the blood, pass through a further developmental stage and then get into the red blood corpuscles, where they grow and become trophozoites. Each mature schizont subdivides into a number of merozoites, which when mature, rupture the red blood corpuscle and become free in the blood-stream (Figs. 33 and 34) Some are engulfed by the white blood corpuscles,

no new ætiological feature is introduced, but exposure and fatigue magnify its effects. Preventive measures and personal protection are frequently difficult, if not impossible, owing to the tactical situation. The introduction of large numbers of non-immunes into endemic areas frequently gives rise to serious epidemics. Normally in peace-time troops are billeted in barracks and cantonments well away from the infected civil and native population, whereas in war the exigencies of the service may necessitate the despatch of troops into the midst of native quarters without it being possible to undertake methods of prevention.

In civilian communities infected individuals can and do treat themselves and can carry on their occupations. The Army, on the other hand, in war-time demands a high standard of physical fitness and ability to go on duty at all times of day or night and in any weather. Thus no medical officer dares to take the responsibility of sending back to duty a man in the acute stages of malaria except in the face of urgent military requirements. Consequently the sick rate of armies exposed to malarial infection is high. In addition men who remain at duty despite malarial infection gradually deteriorate in physical fitness.

Many other factors tend to increase the morbidity from malaria. Infected troops may be moved to a non-malarious area, the local mosquitoes may become infected, and malaria may appear in this previously non-malarial region. Aeroplanes, which can be in England to-day, Africa to-morrow, and back in England the next day, may convey malarial mosquitoes to a non-malarious region. Moreover, troops may be exposed to infection in a highly malarious area for a few days, then return by aeroplane or fast car to a non-malarious area, and when the fever in due course develops malaria may not be suspected. Difficulties in transport of severe cases, difficulties in early diagnosis in advanced and isolated posts, difficulties in early treatment and hospitalisation, all tend to increase the mortality rate in war-time.

Malaria in war-time does not spare the civil population, which may be infected by returning soldiers in places remote from any theatre of war where malaria had almost ceased to exist. Famine, privation and mass migration of refugees may produce malaria in epidemic form in countries where it had been only endemic. In Russia prior to the war of 1914-18 the maximum number of malaria cases per annum was  $3\frac{1}{2}$  millions, in 1923 the number

asia, punctate basophilia and anisocytosis may be seen. The differential count shows an increase in monocytes up to 10-15 per cent. Granules of malaria pigment may be found in mononuclear leucocytes with hyaline cytoplasm and kidney-shaped nucleus.

*Blockage of the capillaries*—This is especially noted in malignant tertian malaria, where the growth and development of the parasite takes place in the internal organs and not in the peripheral circulation. The increase in size of the rings gives rise to alteration in size and shape of the red blood corpuscle, causing it to adhere to the lining wall of the blood capillaries. The corpuscles also have a tendency to adhere to each other causing stasis in the capillary. In cerebral malaria the symptoms are due to the embolism of the cerebral capillaries by agglutinated masses of parasites, pigment and red blood corpuscles. The transient nature of the symptoms is explained by the emboli moving on. A similar process gives rise to the other forms of malignant malaria. In the algid form the suprarenal glands are affected. Pausseau and Lemaire (1916) found that the capillaries of both cortex and medulla contained as many parasites as those of the spleen and more than those of the liver kidneys and other organs. The gland cells showed acute necrosis but hæmorrhage and signs of inflammation were inconstant and comparatively slight. In the choleraic and dysenteric forms the capillaries of the stomach and intestine are plugged with emboli. When the kidneys are affected hæmaturia occurs.

*Pigment*.—Two forms of pigment are elaborated, hæmosoin and hæmoëderin. Hæmosoin is formed by the parasite in the red blood cells. It is liberated into the general blood-stream when the corpuscle ruptures. It is then seized by the phagocytes and transported to the spleen and bone marrow. Hæmoëderin is produced by the breaking down of red blood cells which are uninfected and so is not peculiar to malaria, but is found in many diseases where blood destruction occurs. It is present only in the tissues. The amount of pigment in the body depends on the severity of the attack, but is also influenced by the chronicity of the illness, pigment being eliminated from the tissues as time goes on.

*Spleen and liver*—The spleen is always enlarged. In very acute cases the enlargement may be very slight and the spleen does not become palpable owing to its excessive softness. The colour varies from dark brown to almost black. In the more chronic cases the excessive deposit of pigment causes irritation and fibrosis occurs,

others enter fresh blood cells and start again the non-sexual cycle, others develop into what are called the sexual forms, male and female gametocytes, these are the forms adapted for life in the mosquito. In benign tertian and quartan fever they are spherical, in malignant tertian crescentic. The development of the parasite from sporozoite to the bursting of the mature schizont in the red blood corpuscle and the scattering of merozoites in the blood-stream is called schizogony. The duration of schizogony differs in the various parasites. In quartan it takes 72 hours, in benign tertian 48 hours, and in malignant tertian either 36 to 48 hours. The chill in malaria followed by the fever corresponds with the bursting of the red cell and the flooding of the circulation with the merozoites.

*Mosquito cycle (sporogony)*—When an uninfected female anopheline mosquito bites a person whose blood contains the sexual forms of the malarial parasite, the male and female gametocytes, the blood is sucked into the mosquito's stomach and the development of the malarial parasite in the mosquito begins. The male gametocyte fertilises the female gametocyte and forms an oocyst in the wall of the mosquito's stomach. The oocyst undergoes further development and finally divides into vast numbers of young parasites called sporozoites, which find their way into the salivary glands of the mosquito, which is now infective to man. This cycle (gametogony) usually takes ten to twelve days but may take longer, depending on climatic conditions. The mosquito remains infective for at least one month but may remain infective for the rest of its life if the gut infection was a heavy one. An infected mosquito has been proved to be capable of infecting 15 patients over a period of one month (Shute 1942). When the mosquito bites, the parasites from the salivary glands are injected into the blood and go through the stage of development in the red blood corpuscles described under the human cycle.

### Pathology.

The chief features of the pathology of malaria are anæmia, blockage of the capillaries, deposition of pigment and enlargement of the spleen.

*Anæmia*—The parasite lives in the red blood corpuscle, grows in it and finally destroys it. The reduction in the number of red cells may be considerable. Toxins elaborated by the parasite also play a part in the production of anæmia. The anæmia is of the secondary type with a low colour-index. Achromasia, polychrom-

as the skin becomes moist the temperature falls. It may become subnormal, and the patient feels relieved but exhausted and often sleeps. He may feel quite well after the sweating has ceased and be able to go about his work. Herpes of the lips frequently occurs immediately after the sweating stage, especially in benign tertian fever.

The spleen becomes enlarged to a greater or lesser degree during the attack.

Most attacks start between midnight and noon or in the early afternoon, in marked distinction to fever from liver abscess, tuberculosis, typhoid fever and sepsis, in which the recurrence of fever is usually in the evening. Every day or two there is a fresh paroxysm of the same kind and unless effective treatment is instituted the attacks recur for ten to fourteen days when they get less severe and soon cease for a few days. Spells of a few days of intermittent fever may alternate with spells of freedom from fever for several months during which the patient becomes weaker and more anæmic, and the spleen and often the liver increase in size. Eventually the attacks become less frequent and less severe. Natural recovery takes place, unless the weakened patient is attacked by some intercurrent disease such as pneumonia or dysentery.

The above description of the classical malarial attack applies for the most part to benign tertian and quartan malaria, especially in the relapses. Many variations, however may be noted, especially in malignant tertian and primary benign tertian cases. Rigors may be absent, fever may be indefinite and prodromal symptoms vague. In relapses of benign tertian malaria fever may be remittent at first and later intermittent. In malignant tertian malaria fever is seldom of the classical type. It may simulate typhoid fever without rigors or sweats. Fever may be slight even in severe cases or may be intermittent in daily paroxysms. Splenic enlargement may be difficult to demonstrate. Different geographical strains of the same species vary greatly in virulence and resistance to anti-malarial drugs.

*Special features of benign tertian malaria*—Fever appears every second day (Fig 39). In primary cases the form is usually quotidian. It is seldom fatal, even if no treatment is given. A return of symptoms is very frequent especially with quinine treatment alone. Very few complications occur though bronchitis may develop in the early stages.

*Special features of quartan malaria*—This is much less common

producing the so-called ague-cake spleen. Such a spleen is liable to rupture

The liver is enlarged, but not to the same extent as the spleen. Pigment is found in the Kupffer cells. When much pigment is present the liver becomes slaty-grey to black.

### Symptoms.

The incubation period is usually ten days, but under experimental conditions the mean period after mosquito bite is 14.1 days, after inoculation of malarial blood 11.2 days. In malignant tertian malaria the greater the number of bites, the shorter the incubation period and the more severe the attack. In some cases of B.T. but not in M.T. the incubation period may be as long as 30 to 40 weeks, thus patients who have been in the tropics may develop malaria months after their return to a non-malarial country. The cause of these latent cases is obscure; operations, pregnancy or ill-health may precipitate an attack. If prophylactic quinine is taken, the actual onset of the clinical attack is postponed until the patient has ceased taking the prophylactic drug.

A malarial paroxysm or attack of "ague" consists of a stage of coldness or rigor, a stage of heat, and a stage of sweating. These are followed by a period of apyrexia known as the interval. The duration and intensity of these three classical stages varies considerably. There may be premonitory symptoms of malaise for several days, but more often the onset is sudden, especially in relapses.

The *cold stage* usually lasts one-half to two hours. There is shivering, amounting in many cases to a rigor, the face is pale and pinched, the patient covers himself with everything he can lay hands on, the skin is blue and cold-looking. Vomiting may occur and also headache and aching of limbs. The feeling of cold is entirely subjective. If the temperature is taken, it is found to be several degrees above normal and rapidly rising.

The *hot stage* usually lasts from one to six hours. The shivering abates and gives rise to feelings of warmth growing to intense heat. The headache increases and the temperature rises and may reach 106°. The pulse is rapid, full and bounding. Respiration is hurried. The skin is dry and burning, the face flushed. The clothes are tossed off, and the patient is very thirsty.

The *sweating stage* lasts from two to four hours. The patient generally begins to sweat on the forehead, then all over the body. The sweating is profuse and may even soak the mattress. As soon

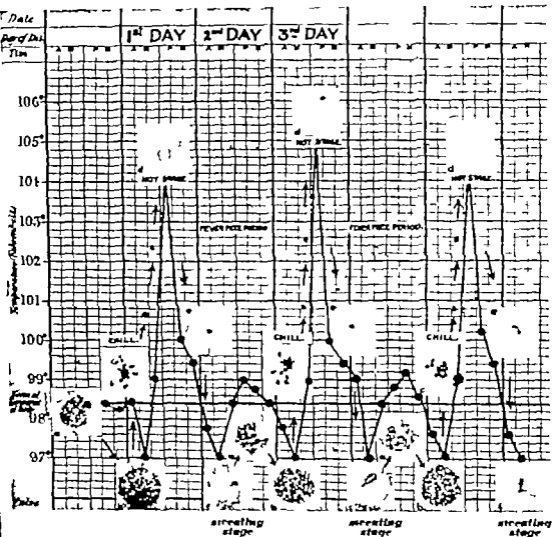


FIG. 30.—Chart illustrating the sequence of clinical stages in benign tertian malaria with the developmental phases of the parasite (*P. vivax*) which correspond to each.

(a) and (f) amoeboid form (b) pre-trophozoite stage (c) rosette body (d) and (e) young schizont.

(Figs. 37, 40, 41, 42, 43, 44, 45, 46, 47, 48, 49, 50, 51, 52, 53, 54, 55, 56, 57, 58, 59, 60, 61, 62, 63, 64, 65, 66, 67, 68, 69, 70, 71, 72, 73, 74, 75, 76, 77, 78, 79, 80, 81, 82, 83, 84, 85, 86, 87, 88, 89, 90, 91, 92, 93, 94, 95, 96, 97, 98, 99, 100, 101, 102, 103, 104, 105, 106, 107, 108, 109, 110, 111, 112, 113, 114, 115, 116, 117, 118, 119, 120, 121, 122, 123, 124, 125, 126, 127, 128, 129, 130, 131, 132, 133, 134, 135, 136, 137, 138, 139, 140, 141, 142, 143, 144, 145, 146, 147, 148, 149, 150, 151, 152, 153, 154, 155, 156, 157, 158, 159, 160, 161, 162, 163, 164, 165, 166, 167, 168, 169, 170, 171, 172, 173, 174, 175, 176, 177, 178, 179, 180, 181, 182, 183, 184, 185, 186, 187, 188, 189, 190, 191, 192, 193, 194, 195, 196, 197, 198, 199, 200, 201, 202, 203, 204, 205, 206, 207, 208, 209, 210, 211, 212, 213, 214, 215, 216, 217, 218, 219, 220, 221, 222, 223, 224, 225, 226, 227, 228, 229, 230, 231, 232, 233, 234, 235, 236, 237, 238, 239, 240, 241, 242, 243, 244, 245, 246, 247, 248, 249, 250, 251, 252, 253, 254, 255, 256, 257, 258, 259, 260, 261, 262, 263, 264, 265, 266, 267, 268, 269, 270, 271, 272, 273, 274, 275, 276, 277, 278, 279, 280, 281, 282, 283, 284, 285, 286, 287, 288, 289, 290, 291, 292, 293, 294, 295, 296, 297, 298, 299, 300, 301, 302, 303, 304, 305, 306, 307, 308, 309, 310, 311, 312, 313, 314, 315, 316, 317, 318, 319, 320, 321, 322, 323, 324, 325, 326, 327, 328, 329, 330, 331, 332, 333, 334, 335, 336, 337, 338, 339, 340, 341, 342, 343, 344, 345, 346, 347, 348, 349, 350, 351, 352, 353, 354, 355, 356, 357, 358, 359, 360, 361, 362, 363, 364, 365, 366, 367, 368, 369, 370, 371, 372, 373, 374, 375, 376, 377, 378, 379, 380, 381, 382, 383, 384, 385, 386, 387, 388, 389, 390, 391, 392, 393, 394, 395, 396, 397, 398, 399, 400, 401, 402, 403, 404, 405, 406, 407, 408, 409, 410, 411, 412, 413, 414, 415, 416, 417, 418, 419, 420, 421, 422, 423, 424, 425, 426, 427, 428, 429, 430, 431, 432, 433, 434, 435, 436, 437, 438, 439, 440, 441, 442, 443, 444, 445, 446, 447, 448, 449, 450, 451, 452, 453, 454, 455, 456, 457, 458, 459, 460, 461, 462, 463, 464, 465, 466, 467, 468, 469, 470, 471, 472, 473, 474, 475, 476, 477, 478, 479, 480, 481, 482, 483, 484, 485, 486, 487, 488, 489, 490, 491, 492, 493, 494, 495, 496, 497, 498, 499, 500, 501, 502, 503, 504, 505, 506, 507, 508, 509, 510, 511, 512, 513, 514, 515, 516, 517, 518, 519, 520, 521, 522, 523, 524, 525, 526, 527, 528, 529, 530, 531, 532, 533, 534, 535, 536, 537, 538, 539, 540, 541, 542, 543, 544, 545, 546, 547, 548, 549, 550, 551, 552, 553, 554, 555, 556, 557, 558, 559, 560, 561, 562, 563, 564, 565, 566, 567, 568, 569, 570, 571, 572, 573, 574, 575, 576, 577, 578, 579, 580, 581, 582, 583, 584, 585, 586, 587, 588, 589, 590, 591, 592, 593, 594, 595, 596, 597, 598, 599, 600, 601, 602, 603, 604, 605, 606, 607, 608, 609, 610, 611, 612, 613, 614, 615, 616, 617, 618, 619, 620, 621, 622, 623, 624, 625, 626, 627, 628, 629, 630, 631, 632, 633, 634, 635, 636, 637, 638, 639, 640, 641, 642, 643, 644, 645, 646, 647, 648, 649, 650, 651, 652, 653, 654, 655, 656, 657, 658, 659, 660, 661, 662, 663, 664, 665, 666, 667, 668, 669, 670, 671, 672, 673, 674, 675, 676, 677, 678, 679, 680, 681, 682, 683, 684, 685, 686, 687, 688, 689, 690, 691, 692, 693, 694, 695, 696, 697, 698, 699, 700, 701, 702, 703, 704, 705, 706, 707, 708, 709, 710, 711, 712, 713, 714, 715, 716, 717, 718, 719, 720, 721, 722, 723, 724, 725, 726, 727, 728, 729, 730, 731, 732, 733, 734, 735, 736, 737, 738, 739, 740, 741, 742, 743, 744, 745, 746, 747, 748, 749, 750, 751, 752, 753, 754, 755, 756, 757, 758, 759, 760, 761, 762, 763, 764, 765, 766, 767, 768, 769, 770, 771, 772, 773, 774, 775, 776, 777, 778, 779, 780, 781, 782, 783, 784, 785, 786, 787, 788, 789, 790, 791, 792, 793, 794, 795, 796, 797, 798, 799, 800, 801, 802, 803, 804, 805, 806, 807, 808, 809, 810, 811, 812, 813, 814, 815, 816, 817, 818, 819, 820, 821, 822, 823, 824, 825, 826, 827, 828, 829, 830, 831, 832, 833, 834, 835, 836, 837, 838, 839, 840, 841, 842, 843, 844, 845, 846, 847, 848, 849, 850, 851, 852, 853, 854, 855, 856, 857, 858, 859, 860, 861, 862, 863, 864, 865, 866, 867, 868, 869, 870, 871, 872, 873, 874, 875, 876, 877, 878, 879, 880, 881, 882, 883, 884, 885, 886, 887, 888, 889, 890, 891, 892, 893, 894, 895, 896, 897, 898, 899, 900, 901, 902, 903, 904, 905, 906, 907, 908, 909, 910, 911, 912, 913, 914, 915, 916, 917, 918, 919, 920, 921, 922, 923, 924, 925, 926, 927, 928, 929, 930, 931, 932, 933, 934, 935, 936, 937, 938, 939, 940, 941, 942, 943, 944, 945, 946, 947, 948, 949, 950, 951, 952, 953, 954, 955, 956, 957, 958, 959, 960, 961, 962, 963, 964, 965, 966, 967, 968, 969, 970, 971, 972, 973, 974, 975, 976, 977, 978, 979, 980, 981, 982, 983, 984, 985, 986, 987, 988, 989, 990, 991, 992, 993, 994, 995, 996, 997, 998, 999, 1000)

than benign tertian or malignant tertian. Fever appears every third day (Fig 40), but, like benign and malignant tertian, in primary cases there is frequently fever every day (quotidian). There is frequently much difficulty in finding the parasites in the blood during the early stages. Paroxysms are longer than in benign tertian. Nephritis is a frequent complication. The mortality is negligible.

*Special features of malignant tertian*—This is by far the most important type of malaria and the most difficult to diagnose, as it seldom conforms to the classical description. No preconceived ideas should be formed as to what the temperature chart in malignant tertian malaria is like. The fever may simulate any disease and frequently suggests typhoid fever, cerebro-spinal meningitis, infective endocarditis or influenza. The main feature is that the patient is usually very ill—much more ill than in benign tertian or quartan malaria, though this may not be apparent and the temperature may not exceed 102°–103° F whereas in benign tertian it may be 105°–107° F. The spleen, though enlarged, may not be palpable. Rigors and sweating may be absent even in fatal cases. Parasites may be extremely difficult to find in the first forty-eight hours, although the patient may be desperately ill. The general symptoms—delirium, vomiting and early jaundice—demand special attention and may be the only feature or features which arouse the suspicion of malaria. The disease is shorter and sharper than benign tertian or quartan fever. Relapses are uncommon. The fever requires prompt treatment by quinine or atabrin, but does not respond as rapidly as the other forms of malaria. The mortality is high if treatment is not promptly instituted. The patient may have trivial symptoms for a few days, then suddenly become delirious or comatose. Thus every case of suspected malignant tertian malaria should be regarded as an urgent emergency. The finding of developing schizonts in the peripheral circulation is of grave prognostic significance, especially if accompanied by rigors.

**Pernicious Attacks.**—These are merely the severe forms of the disease. Attacks have the special feature of coming on with dramatic suddenness, often when least expected. They are frequently fatal. They may supervene on what appears to be an ordinary simple case. They may be the result of lack of prompt treatment or faulty diagnosis. They are usually met with in highly

malarious regions but in these days of rapid travel by aeroplane they may occur in non malarious localities owing to the fact that the patient may have been exposed recently to severe infection while passing through a highly malarious country. The importance of the history in any patient suffering from fever cannot be too strongly emphasised.

Malignant tertian parasites are almost invariably responsible for the grave forms of malaria, but cases have been reported where pernicious attacks have occurred in benign tertian and quartan fever though the possibility of a mixed infection cannot be completely excluded. Pernicious attacks are roughly divided into cerebral, algid, and bilious remittent malaria.

**Cerebral Malaria** (a) *Gradual type*.—This commences with a febrile paroxysm. The gradual onset of coma is generally preceded by premonitory symptoms, such as drowsiness, twitchings, giddiness and slight delirium, but the warning is too frequently ignored. The temperature is variable. It is often high but may be normal.

Recovery may occur after twelve to twenty four hours of coma but death is common, sometimes after a temporary recovery of consciousness.

(b) *Hyperpyrexial type*.—The temperature during the paroxysm may continue to rise till it reaches  $107^{\circ}$  to  $110^{\circ}$ . There may be mania, then coma and death. This type is frequently mistaken for heatstroke.

(c) *Sudden type*.—Sudden coma resembling apoplexy or epilepsy occurs. The temperature is variable. This type is usually fatal in forty-eight hours.

All forms of nervous manifestations may be seen in cerebral malaria, such as epileptiform seizures, tetanic convulsions or symptoms suggestive of meningitis.

**Algid Malaria** (a) *Adynamic type*.—Extreme prostration weakness and collapse occur with a thready pulse cold skin and shallow respirations. The surface temperature may be subnormal, normal or slightly raised, but the rectal temperature is usually elevated. Death is frequent the patient being conscious to the end. In these cases the suprarenal glands are always involved.

(b) *Choleraic type*.—This is similar to the adynamic type, but is associated with rice-water stools simulating cholera.

(c) *Syncopal type*.—Symptoms of cardiac failure are frequently

PLATE VI

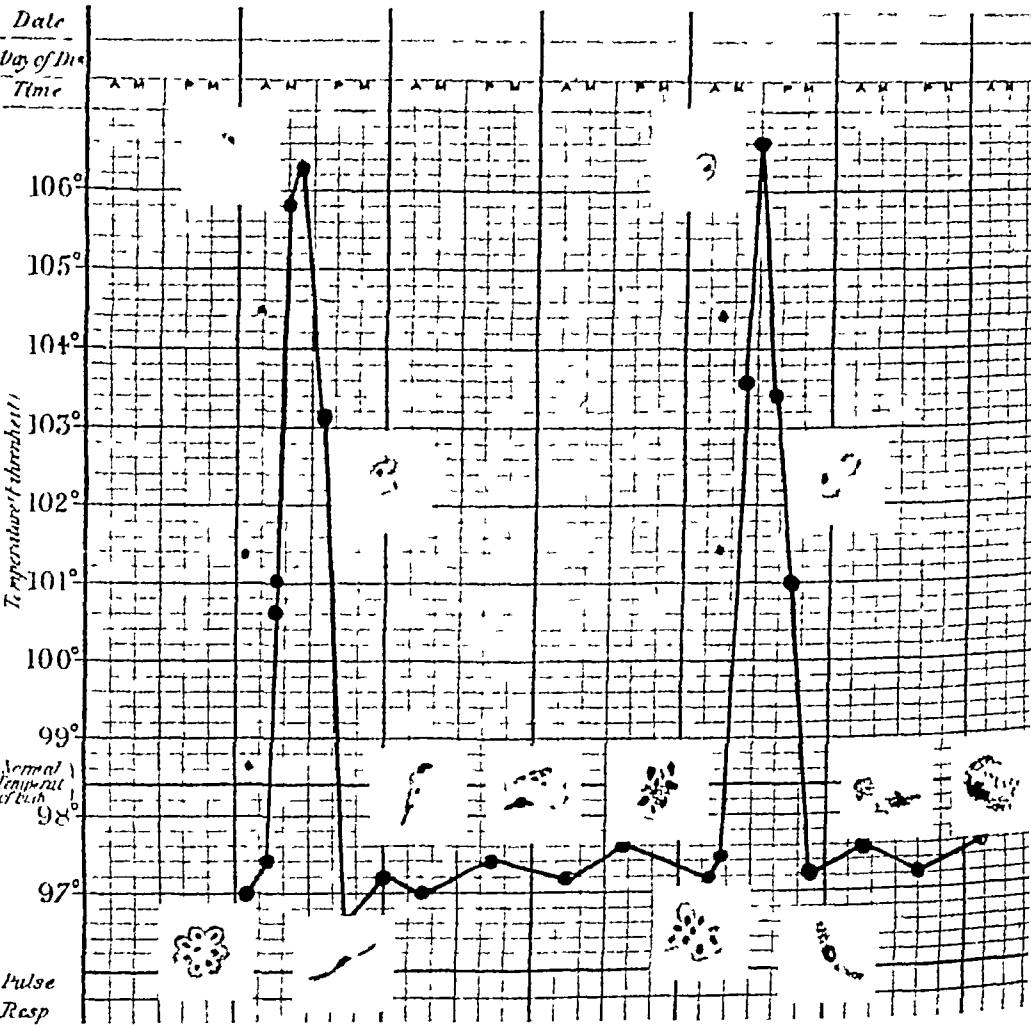


FIG 40 —Chart illustrating the clinical stages in quartan malaria (*P. malariae*) with the developmental phases of the parasite which correspond to them

usually not more than a year from the date of the last exposure to infection.

James (1931) considers that the frequency, duration and severity of relapses depend on the amount of immunity or tolerance a patient may have acquired in the primary attack. Moreover some people may be abnormally susceptible or refractory to infection.

Induced malaria produced by inoculation of malarial blood does not relapse. In untreated primary cases relapses occur in all cases. In patients treated with atabrin and plasmoquin the relapse rate is 11 per cent (Boyd and Amy 1936). In patients treated with quinine plus atabrin and/or plasmoquin the relapses are much less frequent. With plasmoquin and quinine the relapse rate is less than 10 per cent. Malignant tertian relapses less frequently than benign tertian malaria and benign tertian less than quartan.

All forms of relapses conform much more to the classical type of malarial attack than do primary attacks and respond to treatment much more quickly.

### Diagnosis

In any case of acute fever due to malaria the parasite will be found if sought with care and patience. No diagnosis of malaria is reliable without the demonstration of the parasite in a blood smear. In no disease is the necessity for accurate and speedy diagnosis so necessary as in malaria, especially in malignant tertian. A delay of a few hours in giving quinine or atabrin may make the difference between life and death. No fever which does not respond to quinine or atabrin can be malaria. As 90 per cent. of fevers in the tropics are due to malaria, it is essential that a blood smear be taken and examined as soon as a patient comes under medical observation.

Malaria, especially malignant tertian, presents itself in so many different disguises in the tropics and subtropics that failure to exclude malaria by repeated blood smears is malpraxis. The diagnosis of clinical malaria is to be deprecated most strongly. When it is realized that even while under quinine or atabrin treatment malarial parasites can often be found for several days, it will be realized what a confession of laziness the diagnosis of clinical malaria generally is. This does not mean that where a man lacks experience and so fails to find parasites, he should never give anti-malarial drugs. On active service, however in a hyperendemic area without microscopic facilities atabrin or quinine

met with in the pernicious forms of malignant tertian malaria owing to stasis of the capillaries in the heart muscle brought about by infected red blood corpuscles. The heart muscle becomes flabby and œdematous and degenerative changes of a fatty nature develop. Syncopal attacks frequently occur when there is excessive sweating in weak or cachectic patients.

(d) *Dysenteric type*.—In true malarial dysentery the stools are loose and may contain obvious blood, but mucus is usually not present in excess and microscopically there are no pus cells. Many cases of so-called malarial dysentery are, however, caused by a mixed infection with malaria and bacillary dysentery, and the character of the stools is quite different (p. 290).

**Bilious Remittent Malaria.**—Jaundice is present with copious vomiting of bile-stained material. The excess of hæmosiderin produced by the breaking down of red blood corpuscles is said to be responsible for the bilious vomiting. This type may simulate yellow fever.

### Mixed Infections.

Patients may get bitten by mosquitoes carrying benign parasites and at the same time or later by mosquitoes carrying malignant tertian or quartan parasites. Thus one may find in the blood benign tertian and malignant tertian parasites, benign tertian and quartan parasites, or rarely combinations of all three. These mixed infections cause the temperature to vary from the classical picture owing to the different cycles of the parasites. Moreover, one species of parasite may become predominant and the other remain latent, only appearing many months after the attack by the first species is cured. Thus a patient showing quartan parasites only in the blood has been known to develop benign tertian five or six months after the quartan was cured.

### Relapses.

From evidence produced by experimental malaria it appears that trophozoites alone are not concerned in relapses. It is suggested that some of the sporozoites are hidden in the reticulo-endothelial system, where they lie dormant and unaffected by any treatment. The cause of the varying times of the relapses is not clear. In benign tertian relapses seldom occur after one year, but cases have been reported up to two and a half years. In quartan relapses may occur up to twenty years after the original infection. In malignant tertian relapses do not occur after eighteen months and

two to two and a half years after he has left the malarious country is unlikely to be malaria.

No satisfactory serological test is yet available though some experts regard Henry's melano-flocculation test as helpful. Positive Wassermann and Kahn tests may be obtained in patients suffering from malaria if the blood is taken during the acute stage. This positive reaction disappears in a few weeks without treatment. The persistence of a strong reaction should be regarded as evidence of syphilis or yaws.

For the diagnosis from acute and subacute amoebic hepatitis and amoebic abscess of the liver *vide* p 323 and from typhoid fever p 282. In yellow fever severe albuminuria is usual, the fever shorter and pulse slow.

The diagnosis from heat stroke presents great difficulties, as the latter may be clinically indistinguishable from the hyperpyrexial type of malaria. Even if blood smears are negative, intravenous quinine or intramuscular atabrin should be administered at the commencement of treatment.

Kala azar is easily mistaken for malaria in its earlier stages and the fact that the two conditions may coexist adds to the difficulty. An exceptionally low leucocyte count with marked granulocytopenia gives the clue to the possible diagnosis, which can then be confirmed by finding the Leishman Donovan bodies in the material obtained by gland puncture, sternal puncture or splenic puncture.

Malignant tertian malaria may simulate any paroxysmal fever such as meningococcal fever, trench fever (*vide* p 236), undulant fever, infections of the urinary tract, or trypanosomiasis. Laboratory investigations should provide the diagnosis within forty-eight or seventy-two hours in all of these conditions except trench fever.

### Treatment

All our ideas on the treatment of malaria have been based upon quinine and the efficiency of new drugs is judged by comparison with it. Until twelve years ago quinine was the only specific drug available. As a result of the experimental study of therapeutic malaria in general paralysis and the discovery of synthetic anti-malarial drugs the whole position had changed before the outbreak of war in 1939.

One of the most important observations in therapeutic malaria was that the amount of quinine required was much less than had

should be given at once to every case of fever which clinically suggests malaria. But the true diagnosis of malaria is arrived at only by finding parasites in the blood.

In the event of the slide being reported negative further slides should be taken twice daily, one at the height of the fever and one after the temperature has fallen, the patient being carefully watched. No atabrin or quinine should be given unless the case clinically suggests malignant tertian malaria. Then it should be pushed, but the search for parasites should not be discontinued even after the quinine or atabrin has been started. An intermittent temperature which commences to rise in the morning or early afternoon is very suggestive of malaria. A four-hourly chart together with a morning and evening chart should always be kept in any country where malaria is prevalent. The contrast between the ill appearance of the patient one day when his temperature is raised and his comparative fitness on the next when he is apyrexial is seen in no other disease.

An isolated sudden high rise of temperature in an adult is most suggestive of malaria, although similar ague-like attacks may occur in coliform infections of the urinary tract. When malaria is suspected but parasites are difficult to find, the urine should be tested for excess of urobilinogen. If excess is not found it is a strong point against malaria.

Malaria with pain in the abdomen, especially in the right iliac fossa, may easily be mistaken for appendicitis if a blood film has not been examined. During plasmoquin treatment attacks of abdominal pain very like those seen in appendicitis may occur. The history, clinical symptoms and examination of the spleen for tenderness or enlargement give a clue.

In cases where blood smears are negative a differential and total white count should be done. Leucopenia is usual in malaria with a relative increase in the large mononuclear cells, over 15 per cent is significant. Leucocytosis is never present in uncomplicated malaria, it should be an indication for a painstaking search for the presence of another disease even if malaria parasites are found in the film. It may, however, be present during the rigor, but it never persists. One should not be unduly influenced by the history given when a one-time tropical resident who has long since left the tropics complains of "malarial attacks". It should be remembered that any febrile illness occurring in a patient more than

two to two and a half years after he has left the malarious country is unlikely to be malaria

No satisfactory serological test is yet available, though some experts regard Henry's melano-flocculation test as helpful. Positive Wassermann and Kahn tests may be obtained in patients suffering from malaria if the blood is taken during the acute stage. This positive reaction disappears in a few weeks without treatment. The persistence of a strong reaction should be regarded as evidence of syphilis or yaws.

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One of the most important observations in therapeutic malaria was that the amount of quinine required was much less than had

previously been realised. Heroic dosage was found to be unnecessary, wasteful and even harmful

It has long been known that quinine does not act on all the various stages of the malaria parasite, and it has been interesting to find that quinine does not act on the sporozoite. A drug is required which will destroy all stages of the asexual parasites, the sporozoites, schizonts and gametocytes, and which will prevent relapses

Two valuable synthetic drugs have been discovered in recent years. One, atabrin, is schizonticidal, and the other, plasmoquin, is gametocidal. But no one drug has so far been discovered which fulfils all these requirements. Hence in the present stage of our knowledge a combined therapy of quinine, atabrin, and pamaquin is often used with encouraging results (Amy and Boyd, 1936) \*

The loss of 90 per cent of the world's quinine supply to the Allies in 1942 rendered the position as regards malaria critical and stimulated the search for substitutes. Many new drugs have been tried, including the sulphonamide group, but these trials are still in the experimental stage, though sulphadiazine has given promising results in the treatment of M T malaria. Further attention has been paid to the possibilities of atabrin, and there is increasing evidence that this drug is an excellent substitute for quinine and in many ways may prove to be its superior, especially in M T infection. At present it is the only valuable substitute for quinine. Theoretically the treatment of malaria depends on a large number of factors, all of which should be considered before embarking on any course of therapy. Among these are the degree of natural susceptibility or resistance, the degree of tolerance or immunity which has been acquired before treatment, the species of parasite concerned, and the virulence of the particular geographical strain of the parasite.

In war-time especially, when one has to deal with large numbers

\* Atabrin and plasmoquin, both Bayer products imported from Germany, are now being produced in England under the pharmacological names of mepacrine hydrochloride and pamaquin. These English products have been passed by the Medical Research Council as having equivalent properties to the imported proprietary preparations. In this chapter the original trade names have been retained, as they are so well known to the medical profession. Quinacrine and praequine, the French equivalents of atabrin and plasmoquin, are now being made in England by May & Baker.

of cases, it is necessary to have certain standard methods of treatment, which can be modified to suit individual cases. The work of Amy and Boyd (1936) in dealing with the malarial problem in the whole of the British Army in India, both British and Indian troops has shown the efficiency of this standard method of treatment, especially in preventing relapses.

Theoretically no patient should ever die of malaria for in atabrin and quinine we have specific remedies which if given in time, will invariably cure the disease, although there may be a certain number of relapses. Practically patients with malaria do die and die in thousands every year. Of malaria patients who died in Salonica in 1917 57 per cent. died forty-eight hours after admission to hospital mainly owing to failure of the patient to report sick sufficiently soon, and to failure of the medical officer to realise that speed in diagnosis and treatment is essential.

The patient should be sent to bed at once and remain there for at least five days after the temperature has been normal. Powders of 5 gr. aspirin, 5 gr. phenacetin and 2 gr. caffeine relieve the headache, aches and pains, but have no curative effect on malaria. Atabrin and quinine are the only drugs that have any antipyretic effect on malaria. Tepid sponging is very useful when the temperature is raised and the patient cannot sleep. In hyperpyrexial cases cold sponging may be necessary. All anti-malarial drugs are best taken after meals when twice daily after breakfast and after tea, and when thrice daily the last dose after dinner or supper. Each dose of atabrin, quinine or plasmoquin may be preceded by a dose of alkaline mixture, containing 20 gr. each of sodium bicarbonate and potassium citrate. A plain enema should be given at the onset of the attack and the bowels kept open by magnesium or sodium sulphate when necessary. This is especially important when atabrin or plasmoquin is being given.

The diet should be fluid while the temperature is raised but a full generous diet is necessary in the apyrexial periods and in convalescence. Hot sweet tea is especially useful when large doses of atabrin are being taken.

**Atabrin**—Atabrin is available as tablets of 0.1 grm. for oral administration and as atabrin musonate (mapacrine dimethane sulphonate) in ampoules containing 0.375 grm. equivalent to 0.3 grm. of atabrin, for intramuscular injection. Atabrin has no toxic effects, but if taken for long periods produces a harmless

yellow discoloration of the skin, especially those parts exposed to the air. Only in rare cases does it stain the conjunctivæ yellow. Atebrin should not be given in combination with plasmoquin. Two days' rest must be allowed between atebrin and plasmoquin courses. It can, however, be given at the same time as quinine, but this is seldom necessary. A single dose of 0.6 gm atebrin has a slightly more rapid action on the trophozoites of *P. malariae* and *P. vivax* than 15 or even 20 grains of quinine, and freedom from relapses lasts longer. A single dose of 0.6 gm atebrin produces well-marked changes in the blood, which begin in about half an hour and can best be studied in unstained films. The pigment which is scattered through the cytoplasm of the fully grown B T schizont clumps into one or two masses, and two or three hours later many parasites have completely lost their pigment. Still later disintegration of the cytoplasm is seen. If the atebrin is given in adequate doses at the beginning of a primary attack, gametocytes will probably not be formed, and if they do appear they will probably be scanty in all species if the attack is treated early (Shute, 1943). Atebrin acts more rapidly on the trophozoites of *P. falciparum* than quinine, especially in strains which are quinine resistant. The experiments of Nicol and Shute strongly suggest that atebrin also acts on the sporozoites of M T (*vide p* 381). It is frequently stated that atebrin does not control the temperature as rapidly as quinine, probably because the dose of 0.3 gm daily for five days formerly in use was insufficient.

Most cases of malaria respond to the following treatment: 0.6 gm atebrin in a single dose, followed by 0.3 gm daily for the next six days, 2.1 to 2.4 gm being the usual amount required to treat a case of B T or M T. If, however, fever is severe and parasites numerous on the third or fourth day of treatment, the next dose can be safely increased from 0.3 to 0.6 gm. Recent work has shown that 0.6 gm daily over a period of six days can be given when necessary.

Atebrin musonate is put up in ampoules containing a yellow sterile powder, which should be dissolved in 7½ to 10 c cm of distilled water and injected into a muscle.

The injection is generally painless. A very good preparation suitable for intramuscular or intravenous injection is atebrin dihydrochloride (Wintrop), which is supplied in ampoules containing 0.2 gm of the sterile powder. To facilitate the prepara-

tion of fresh solutions for urgent cases, a 10 c.cm. ampoule of distilled water is supplied with each dose. The indications for intramuscular atabrin are the same as for intravenous quinine.

The intravenous injection of atabrin is seldom necessary as it has no advantage over the intramuscular route which is the method of choice when oral administration is not possible.

Atabrin does not provoke an idiosyncrasy like quinine. It is not contraindicated in the onset or threatened onset of blackwater fever. A constant bitter taste and some depression may be noted with large doses of atabrin and the urine becomes bright yellow. Nausea indicates the necessity for discontinuing or reducing the dose which can be done at any time during the course provided the fever is under control. Failure to become yellow is not necessarily evidence that a patient is failing to absorb the drug as many people do not become yellow although absorption is satisfactory. The tablets of atabrin can be swallowed whole and are absorbed. Crushing the tablets and giving the drug in powder form produces an extremely bitter taste in the mouth, which becomes stained a greenish yellow.

Prophylactic atabrin up to 0.1 grm. daily does not cause nausea or gastro-intestinal symptoms.

*Test for atabrin in the urine.*—10 c.c. urine are rendered alkaline in test-tube with a few drops of sodium hydroxide. 0.25 c.c. amyl alcohol is added and well shaken. On separation atabrin is seen in the upper layer of amyl alcohol as a yellow colouration when the test tube is viewed by transmitted light against dark background. Atabrin is excreted slowly and does not normally appear in the urine until 1.4 grm. have been given. Too rapid excretion in the urine means that the necessary blood level is not being maintained.

*Plasmoquin*.—Plasmoquin is available in tablets of 0.01 grm. suitable for oral administration. It should not be given intravenously. It has no action on the trophozoites of *P. falciparum* slight on those of *P. vivax* and considerably more on those of *P. malariae*. Small doses given with quinine appear to reinforce its action on the trophozoites of *P. vivax* and *P. falciparum*. It acts on gametocytes of all species equally but because gametocytes of M.T. are not affected by quinine plasmoquin is especially valuable against this species. In association with quinine or following atabrin it is believed to reduce the number of relapses of all three

types. The two- or three-week method of treating B T malaria with 20 grains of quinine and 0.03 gm of plasmoquin daily, which was standard in India some years ago, materially reduced relapses, but has fallen into disuse recently, owing to the large amount of quinine required and the long period of hospitalisation. I treated six hundred cases by this method with excellent results. There were no toxic symptoms and the relapse rate was less than 4 per cent as compared with 25 per cent when only quinine was given (Dixon, 1933). It was believed that the toxicity of plasmoquin was reduced when given in combination with quinine. The toxic symptoms of plasmoquin given in reasonably small doses, 0.02–0.03 daily, has been much exaggerated, but abdominal pains and a mild degree of cyanosis may occur. Cessation of treatment for twenty-four hours relieves both. Barley sugar or glucose should be given while the patient is on plasmoquin treatment, the bowels kept open with sodium sulphate, and no strenuous exercise should be taken.

Plasmoquin should be given only under medical supervision and should be administered only by mouth. The modern method is to give plasmoquin after a short course of quinine or atabrin when the acute stage of the malarial attack has subsided and then only for five days in doses not larger than 0.03 gm daily. Patients should be kept in hospital when plasmoquin, 0.03 gm, is being given daily, but those carrying crescents on discharge from hospital can take 0.02 gm twice weekly without any ill effects. This treatment may be valuable, because new crops of crescents are sterilised even if they appear in the peripheral blood some days after leaving the hospital.

*Quinine*—Quinine acts on the trophozoites of *P. vivax*, *P. malariae* and *P. falciparum* and is an effective schizonticide in all the forms of malaria. In B T infections, gametocytes disappear from the blood following quinine treatment. The period which elapses between giving quinine and the disappearance of gametocytes is usually three to five days, but so long as they are present in sufficient numbers, they are infective to mosquitoes. In M T, however, quinine has little or no effect in clearing the gametocytes from the blood, and this is where plasmoquin fulfils a very useful purpose because it sterilises the sexual forms. Quinine does not cure the disease nor does it always prevent relapses. If given in doses of from 10 to 30 grains daily for not more than seven days,

it does not hinder the process of immunisation. Large doses may be required in quinine resistant strains.

Vomiting after quinine is caused by the malaria, and nothing cures it quicker than quinine or atabrin. If vomiting occurs within half an hour more should be given, as it probably will be retained. An ounce and a half of the following mixture given every hour is also useful in controlling persistent vomiting, which usually ceases after the second dose.

Sodium bicarbonate	150 gr
Mercury perchloride	$\frac{1}{2}$ gr
Water to	1 quart

Tablets of quinine dihydrochloride are frequently retained when solutions are vomited. Atabrin may be substituted if vomiting persists. As a last resource quinine must be given intravenously.

A moderate degree of ringing in the ears and temporary deafness are inevitable in treatment with quinine. They serve a useful purpose by showing that the quinine is being absorbed. In the doses recommended excessive cinchonism is unlikely to occur. Quinine bihydrochloride, which is soluble in 0.6 parts of water is the salt of choice because of its extreme solubility. It may also be given in tablet form or in cachets. Quinine bisulphate, soluble in 10 parts of water is second choice on account of solubility. Tablets, pills or cachets of the other salts should never be used, as they are likely to pass through the intestines unchanged.

In cases of malaria which do not react to quinine it is always possible that the patient may not be taking the dose prescribed owing to the unpleasant taste, or it may not be absorbed although it has been swallowed. Howie and Murray Lyon (1943) found that seven out of a series of a hundred patients with malaria taking quinine orally failed to excrete quinine until it was given intravenously.

It is the custom in the Italian army to impregnate quinine tablets with methylene blue. This shows whether the quinine is being taken, but it does not prove that it is being absorbed. Mayer Tanret's test affords a ready and simple proof that quinine is being absorbed. It gives a positive reaction in a dilution of 1 in 1 200 000. It is best carried out between thirty and ninety minutes of taking the quinine.

The Mayer Tanret test should be carried out in all cases of malaria which fail to respond to quinine quickly and in all cases of severe

types. The two- or three-week method of treating B T malaria with 20 grains of quinine and 0.03 gm of plasmoquin daily, which was standard in India some years ago, materially reduced relapses, but has fallen into disuse recently, owing to the large amount of quinine required and the long period of hospitalisation. I treated six hundred cases by this method with excellent results. There were no toxic symptoms and the relapse rate was less than 4 per cent as compared with 25 per cent when only quinine was given (Dixon, 1933). It was believed that the toxicity of plasmoquin was reduced when given in combination with quinine. The toxic symptoms of plasmoquin given in reasonably small doses, 0.02–0.03 daily, has been much exaggerated, but abdominal pains and a mild degree of cyanosis may occur. Cessation of treatment for twenty-four hours relieves both. Barley sugar or glucose should be given while the patient is on plasmoquin treatment, the bowels kept open with sodium sulphate, and no strenuous exercise should be taken.

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soluble salts being absorbed as quickly as soluble provided they are given in solution. The rate of absorption varies according to the condition of the gastric and intestinal mucosa, but usually absorption is complete within six hours.

The amount of quinine excreted by the urine gives the best indication of the effect produced by the drug, and it is obviously unnecessary to give either intravenous or intramuscular quinine if the patient's urine shows good absorption when quinine is given by mouth. The excretion is the same by whatever route it is administered. It usually appears in the urine within thirty to ninety minutes, but it may be delayed as much as five hours. The excretion in the urine reaches its maximum in six to nine hours. If it does not appear in the urine within six hours it may be assumed that it is not being absorbed. When very large doses are given there is little increase in the amount excreted as compared with moderate doses. The surplus is deposited in the liver spleen and other organs in a useless or even harmful form. Quinine given either by the mouth or intravenously remains in the blood a very short time.

*Routine treatment of malaria*—The following is the best routine treatment for all B.T. quartan and mild M.T. infection.

*1st day*—Atebrin 0.2 grm. every four hours until 0.6 grm. is given. It is important to get a high concentration of atebrin in the blood during the first twelve hours.

*2nd-7th day inclusive*.—Atebrin 0.1 grm. three times a day.

*8th and 9th day*—No anti-malarial treatment.

*10th-14th day*—Plasmoquin, 0.01 grm. three times a day.

Thereafter atebrin 0.01 grm. daily all the time the patient remains in the malarious area and for thirty days after leaving it.

When 0.6 grm. atebrin in the first twelve hours does not control the fever it should be continued in the dosage of 0.2 grm. three times a day for a further twenty-four hours. If this does not control the temperature, intramuscular injection of atebrin musonate 0.3 grm. should be given and repeated if necessary in twelve hours. It should then be possible to resume the routine treatment recommended above as from the fifth day, i.e. a further two days of atebrin, 0.3 grm. orally then no treatment for two days, followed by plasmoquin.

*Alternative method of treatment if atebrin is not available*.—Quinine bihydrochloride, gr 10 three times a day for seven days, com

M.T. malaria on the first and second day It should also be done occasionally when prophylactic quinine is being given to ensure that the dose is being taken and absorbed.

*Mayer Tanret's quinine absorption test*

- (1) Test the urine for albumin; if this is absent, add 6 to 7 drops of Tanret's solution to 2 c cm of filtered urine This should give a dense white precipitate, soluble on heating, if quinine is present in ordinary amount
- (2) If urine contains albumin, add Tanret's solution and boil Filter while still hot As the filtrate cools, the appearance of a precipitate soluble on further heating indicates the presence of quinine The amount of quinine per ounce of urine can be calculated by measuring with Brown's tubes

Composition of Tanret's solution

Potassium iodide	3 grm
Corrosive sublimate	1 grm
Glacial acetic acid	20 c cm
Distilled water to	60 c cm

The indications for the intravenous injection of quinine are coma in pernicious malignant tertian cases, and failure to absorb quinine owing to persistent vomiting in bilious remittent and other severe malignant tertian cases, such as the dysenteric and hyperpyrexial forms The advantages of intravenous quinine are its speed and certainty of absorption It should be reserved for cases of extreme urgency If the patient can swallow and is absorbing quinine by mouth, there is no justification for giving it intravenously Its danger is the fall of blood pressure which follows the injection and may cause collapse or syncope In the very rare cases of quinine intolerance death may follow Bihydrochloride of quinine should be used in doses of  $7\frac{1}{2}$  grains dissolved in 10 c cm of distilled water,  $\frac{1}{2}$  c cm of pituitrin may be added to the solution to prevent collapse In algid or collapsed cases half to one pint of saline and 5 per cent glucose solution should be added The solution is boiled in a test tube and injected into the medial basilic vein The injection should be made slowly, at least three minutes being allowed for the 10 c cm.

There are no indications for subcutaneous, intramuscular and rectal injection They are all dangerous and have now been almost universally given up

When given in solution or after being dissolved in gastric juice quinine is rapidly absorbed, chiefly from the small intestine, less

soluble salts being absorbed as quickly as soluble provided they are given in solution. The rate of absorption varies according to the condition of the gastric and intestinal mucosa, but usually absorption is complete within six hours.

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Thereafter atebrin 0.01 gm. daily all the time the patient remains in the malarious area and for thirty days after leaving it

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bined with plasmoquin 0.01 grm for the last five days of the course. If the fever is not controlled after forty-eight hours of this treatment, quinine, gr  $7\frac{1}{2}$ , should be given intravenously and repeated if necessary in twelve hours, after which oral medication should be possible. The quinine absorption test should be made on the first two days in all cases of malignant tertian malaria and in any case which does not rapidly respond to oral quinine. Failure to absorb oral quinine is an immediate indication for intravenous quinine.

For cerebral malaria, coma, hyperpyrexia, bilious remittent, or any severe malarial attack in which the patient is unable to swallow, retain or absorb anti-malarial drugs, quinine, gr  $7\frac{1}{2}$ , should be injected intravenously and repeated in eight hours and/or until the patient is able to take quinine by the mouth, or intramuscular atebryn musonate 0.6 grm repeated if necessary in twelve hours, and thereafter at twenty-four-hour intervals until the above routine treatment can be started. Usually one intravenous injection of quinine or one intramuscular injection of atebryn musonate is sufficient to permit oral medication.

*Cerebral malaria* — Quinine should be given intravenously at the earliest possible moment. Usually one injection is sufficient, but if necessary the injection may be repeated in six to eight hours. The normal symptomatic treatment for coma should be carried out—ice-bags to the head, hot applications to the feet and legs. If respiratory distress is present oxygen should be given. Hyperpyrexial cases require cold packs and ice enemata, these should be continued till the rectal temperature falls to  $102^{\circ}$ .

Lumbar puncture followed by withdrawal of cerebro-spinal fluid if the pressure is raised is useful in those cases in which there is evidence of raised intra-cranial pressure due to cerebral cedema, and should always be carried out if the patient does not react to the intravenous quinine.

**Prophylactic or Suppressive Treatment by Quinine or Atebryn** The dose of quinine usually recommended for prophylactic or suppressive treatment is 5 grains daily. That quinine, even in relatively large doses, such as 30 grains daily, is not a true causal prophylaxis may be seen in cases where the drug is given for seven or eight days of the incubation period and then stopped. In such cases fever begins two or three days later, but parasites may be scanty for some days after fever begins. If, however, the

daily dose of quinine is continued beyond the incubation period, there is often slight fever within the usual incubation period but it soon terminates. The reason for this is that the quinine given at the end of the incubation period is acting on the parasites, which have by this time appeared in the blood-stream in sufficient numbers to cause fever.

A true causal prophylactic in malaria is affected by a drug which prevents the infecting organisms or sporozoites from surviving long enough to cause clinical symptoms appear as trophozoites in the blood-stream, cause short- or long-term relapses, or a fall in the haemoglobin percentage sufficient to affect the well being of the infected person.

When atabrin is available quinine is not recommended as a prophylactic. Atabrin is the drug of choice for mass prophylaxis, and contrary to what was previously thought it can be given without medical supervision. It should be given in doses of 0.1 gm. after meals six days a week, excluding Sundays. Although there is evidence to show that 0.2 gm. twice a week is sufficient there is less likelihood of doses being missed if it is taken each week-day omitting Sunday only. It should be commenced at least two weeks before the patient enters a malarious locality and should be continued for at least thirty days after leaving.

Causes of failure in atabrin prophylaxis—Shute (1943) defines causal prophylactic as a drug which while circulating freely in the blood during the preinoculation period, will prevent malaria developing parasitologically and clinically. The efficacy of atabrin as a causal prophylactic in M.T. malaria appears to depend on the maintenance of an undetermined blood level of the drug at all times when exposed to infection. Failure in atabrin prophylaxis is probably due to the concentration of the drug in the blood being too low. This may be caused by a fluctuating blood level through neglect to take the maintenance dose of 0.1 gramme daily or the drug may be excreted too rapidly thus preventing the necessary blood level being reached and maintained, owing to individual idiosyncrasy or special conditions producing excessive sweating and large intake of fluid. When it is known that the drug is being excreted rapidly by profuse sweating or through the kidneys readjusting themselves to the changed conditions, it may be advisable to increase the daily dose of prophylactic atabrin from 0.1 gm. to 0.2 gm.

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out by James and Shute in 1934 at the Devon Mental Hospital, is an illustration of this.

More recent trials with atabrin against the malignant tertian parasite suggests that even in very small doses atabrin may be a true causal prophylactic, i.e. that the infecting organisms (sporo-

### BENIGN TERTIAN MALARIA (*P. vivax*)

#### Prophylactic trials

Prophylactic Doses.	No. of Cases.	How Infected.	Result within Usual Incubation Period.	Final Results.
Quinine, 45 gr., on the evening before infection and the same dose next day at actual time of infection.	3	Mosquitoes	All developed fever and parasites within 15 days.	Two had recurrences 8 and 11 months after infection.
Atabrin, 0.5 grm. on the evening before infection and the same dose next day at actual time of infection.	3	Mosquitoes.	No attacks.	All developed acute attacks 9-12 months after infection.

### MALIGNANT TERTIAN MALARIA (*P. falciparum*)

#### Prophylactic trials

Prophylactic Doses	No. of Cases.	How Infected.	Period of Observation.	Final Results
Quinine, 15 gr., daily for 7 days beginning day before infection.	4	Mosquitoes.	8 years.	All developed fever and parasites within 18 days.
Atabrin, 0.3 grm., daily for 6-7 days beginning day before infection.	13	Mosquitoes.	8 years.	No parasites and no fever throughout.

zoites) are destroyed before they succeed in attacking the red blood cells providing that the concentration of the drug in the blood is at the requisite level at the time of the infection.

The following table illustrates this. It is the result of some trials recently carried out by Nicol and Shute (1943) at Horton Hospital.

In the event of the malaria breaking through the "atebrin barrier" the resulting malaria attack is just as severe as if no prophylactic atebtrin had been taken. Parasites are numerous and the fever is severe, at least in primary cases. In deciding on the treatment of such an attack, no allowance should be made because the patient has been taking small daily doses of atebtrin over a long period (Shute, 1943)

Quinine is a recognised suppressive drug in malaria, but in the present state of our knowledge it is doubtful whether atebtrin, 0.1 gm daily, acts as a suppressive drug in primary M T malaria. The experiments of Nicol and Shute, tabulated below, show, however, that atebtrin prolongs the incubation period in B T. malaria.

It is also doubtful whether atebtrin, 0.1 gm daily, acts as an anti-relapse treatment in cases which, as the results of an acute attack of malaria, have been compelled to go into hospital or have had to receive therapeutic treatment elsewhere. If the small dose of 0.1 gm atebtrin is started again following discharge from hospital, it will protect against reinfections, but it will not necessarily prevent relapses. The best way of preventing relapses is early diagnosis and thorough treatment with atebtrin in sufficiently large doses.

It is well known that tolerance to one or two strains of M T malaria is quickly acquired by the individual, at least for a time. Therefore care must be taken not to conclude hastily that where parasite carriers are discovered, who are taking 0.1 gm. of atebtrin daily, the drug is acting as a suppressive, as it may be because the patient is immune.

The experiments summarised below are very interesting and indicate that atebtrin is a causal prophylactic in M T. In B T infections atebtrin in similar doses prevents the onset of the primary attack for periods of six months to one year.

There is much evidence to show that atebtrin has some advantages over quinine and pamaquin both as a suppressive anti-malarial drug and as a causal prophylactic drug, especially with the species of malarial parasite which causes malignant tertian fever. If 0.3 gm of atebtrin is given on the day before infection with malaria and continued for a few days after infection, no attack will ever occur, thus showing that atebtrin is a useful prophylactic drug. The following table, which summarises the results of trials carried

and characterised by pyrexia, hæmoglobinuria, bilious vomiting and jaundice with rigors and frequently diminution and suppression of urine. The onset is sudden and explosive the hæmolysis of the red blood corpuscles appears to take place within a few hours, and the rest of the illness consists of the after-effects of the hæmolysis. It occurs in places where severe malignant tertian malaria persists throughout the greater part of the year. It is commonest amongst Europeans, and usually occurs among those who have had several imperfectly treated attacks of malignant tertian malaria. *Plasmodium falciparum* parasites are present in almost 90 per cent. of the cases.

#### Ætiology

In certain unknown conditions the red blood corpuscles seem to become sensitised to the action of the hæmolyxin produced by the action of quinine on the malarial parasites. The sensitised corpuscles become dissolved when a fresh supply of hæmolyxin is brought into existence by the action of quinine on a new brood of parasites or by the parasites themselves. There is no evidence of quinine idiosyncrasy nor excessive fragility of the red blood corpuscles.

#### Symptoms

The illness usually begins with a rigor. There is then pain in the loins and an urgent desire to micturate. The urine is at first pinkish, then red and finally port-wine or even a much darker porter colour for from a few hours to two days. The temperature is invariably raised to 103° to 105° falling as the urine clears. Within a few hours jaundice appears with vomiting and nausea. The liver and spleen become enlarged and tender. Anæmia develops rapidly as many as 2 000 000 red cells per cubic centimetre may be destroyed in twenty four hours.

In mild cases the urine clears, the temperature falls the patient sweats, and the symptoms disappear. Such cases may be missed unless the urine is examined carefully. In severe cases the symptoms increase in severity. Hiccup is present, anuria develops, thirst is extreme and the patient may die from cardiac failure, anuria, hyperpyrexia, or anoxæmia from insufficiency of red cells to carry the necessary oxygen.

#### Diagnosis

The patient has usually made the diagnosis himself before the doctor arrives. He has been living in a malarious area for some

MALIGNANT TERTIAN MALARIA (*P. falciparum*)

## Atebrin (Mepacrine) Causal Prophylaxis

Prophylactic Doses	No of Cases	How Infected	Remarks	Results
0 1 grm daily except Sundays starting 22 days before first infection and continuing for 30 days after last infection in one patient, and 60 days in the other	2	Mosquitoes	Infected twice weekly over a period of 3 weeks	No parasites or fever Under observation to date, 6 months
0 2 grm Saturdays and Sundays Beginning and ending as for above	—	Mosquitoes	Infected twice weekly over a period of 3 weeks	No parasites or fever Under observation to date, 6 months.

Some interesting points connected with these later trials are

- 1 There was no drop in the hæmoglobin throughout the experiment
- 2 The mosquitoes used were very heavily infected
- 3 Although the patients took the drug over a long period of time, in two cases for over three months, there was only slight discoloration of the skin and then only on the exposed parts of the body
- 4 The patients were up and about and working

The importance of these latest trials is obvious. It indicates that troops and ships' crews may remain free from malaria over a long period of time even in highly endemic areas, providing they take atebrin regularly. In estimating the value of this drug as a causal prophylaxis in the field care should be taken to include only those people who have never been infected previously, or for at least two years before. We do not yet know whether the small quantities mentioned above are sufficient to prevent relapses.

## BLACKWATER FEVER

The hæmoglobinuria which results from malignant tertian malaria has in the past been regarded as a separate entity. It is, however, really a complication of a severe attack of malignant tertian malaria. All grades of malarial hæmoglobinuria are of similar origin, the degree of severity varies, the most severe being known as blackwater fever. It is an acute illness produced by malarial infection.

and characterised by pyrexia, hæmoglobinuria bilious vomiting and jaundice with rigors and frequently diminution and suppression of urine. The onset is sudden and explosive the hæmolytic of the red blood corpuscles appears to take place within a few hours, and the rest of the illness consists of the after-effects of the hæmolytic. It occurs in places where severe malignant tertian malaria persists throughout the greater part of the year. It is commonest amongst Europeans, and usually occurs among those who have had several imperfectly treated attacks of malignant tertian malaria. *Plasmodium falciparum* parasites are present in almost 90 per cent. of the cases.

### Ætiology

In certain unknown conditions the red blood corpuscles seem to become sensitised to the action of the hæmolytic produced by the action of quinine on the malarial parasites. The sensitised corpuscles become dissolved when a fresh supply of hæmolytic is brought into existence by the action of quinine on a new brood of parasites or by the parasites themselves. There is no evidence of quinine idiosyncrasy nor excessive fragility of the red blood corpuscles.

### Symptoms

The illness usually begins with a rigor. There is then pain in the loins and an urgent desire to micturate. The urine is at first pinkish, then red and finally port-wine or even a much darker porter colour for from a few hours to two days. The temperature is invariably raised to 103° to 105° falling as the urine clears. Within a few hours jaundice appears with vomiting and nausea. The liver and spleen become enlarged and tender. Anæmia develops rapidly as many as 2 000 000 red cells per cubic centimetre may be destroyed in twenty four hours.

In mild cases the urine clears, the temperature falls the patient sweats, and the symptoms disappear. Such cases may be missed unless the urine is examined carefully. In severe cases the symptoms increase in severity. Hiccup is present, anuria develops, thirst is extreme and the patient may die from cardiac failure anuria, hyperpyrexia, or anoxæmia from insufficiency of red cells to carry the necessary oxygen.

### Diagnosis

The patient has usually made the diagnosis himself before the doctor arrives. He has been living in a malarious area for some

time and has had several previous attacks of malaria. The fever has come on suddenly with a rigor and vomiting. The urine is red or black in colour and jaundice soon appears. Sometimes the patient may have left the malarious area and is living in a temperate zone. The diagnosis has then to be made from the following conditions.

*Hæmaturia* —In blackwater fever the urine contains no red blood corpuscles.

*Bilious remittent fever* —This may have some clinical resemblance, but examination of the urine will clear up the diagnosis. If a spectroscope is not available, filter paper may be used to show the difference between bile and hæmoglobin in the urine. If bile is present, as in bilious remittent fever, the filter paper turns yellow, if hæmoglobin is present the filter paper becomes red.

*Paroxysmal hæmoglobinuria* —Fever and rigor are less pronounced, vomiting is usually absent, and there is usually a clear history of previous attacks, which have occurred under conditions in which blackwater fever can be excluded.

*Spirochaetal jaundice and yellow fever* —Hæmoglobinuria is rare in these diseases, jaundice develops later, and bile invariably is found in the urine.

### Prognosis.

The prognosis depends on the severity of the attack, the previous health of the patient, and the treatment. The mortality varies from 10 to 40 per cent. Severe rigors, hyperpyrexia, anuria, deep jaundice, recurrence of the paroxysms, hiccups and profound anæmia are all unfavourable features.

### Treatment.

The patient must be kept warm in bed and not moved. Good nursing is the most important factor in recovery.

Symptomatic treatment is mainly required. By the destruction of the red cells the body has rid itself of the malaria. There is therefore seldom any necessity for anti-malarial treatment during the acute stages. The modern view is that there is the same lesion of the kidneys as in a mismatched blood transfusion and the crush syndrome, the degree of anæmia depending on the amount of hæmolysis.

For milder cases large quantities of bland fluids should be given with sufficient sodium citrate to lessen the clogging of renal lobules with debris and acid hæmatin and thus prevent anuria. For

severe hæmoglobinuria or persistent vomiting 150 c.cm. of 3 per cent solution of sodium citrate should be given intravenously and followed by a continuous drip for twenty four hours of a further 450 c.cm. of citrate and 2,400 c.cm. of 5 per cent glucose solution. If no further hæmolysis has occurred this should have cleared the kidneys and unless the blood count is below two million a blood transfusion is contraindicated. If the red-cell count is one million or below or further hæmolysis is occurring, it is necessary to give blood. A suitable donor should be in readiness in every case of blackwater fever. He should be tested for malaria. The possibility of the fresh blood complicating the hæmolysis in the patient should be borne in mind—this is the reason for avoiding transfusion of blood as far as possible. Should the red-cell count drop to less than one million per cubic millimetre there is a danger of pulmonary œdema, and at counts of  $1\frac{1}{2}$  million or less it becomes imperative to transfuse.

Dry cupping of the loins may promote the flow of urine. Intravenous injection of anti-venenes has been used with success. After the acute stage is passed the blood should be examined for malarial parasites, which usually appear five to fourteen days after the subsidence of the hæmoglobinuria. As soon as they appear a course of atabrin should be given as for malignant tertian malaria, but not plasmoquin, which itself predisposes to methemoglobinæmia. Patients who have had blackwater fever should keep away from malarious localities for the rest of their lives, as one attack predisposes to recurrences.

### BLOOD SMEAR TECHNIQUE

Every practitioner should be able to take a blood smear and know how to recognise malarial parasites in thin blood films.

Use only clean and well polished slides. Frosted and scratched slides are useless. Store the slides in rectified spirit in a glass jar with a ground glass stopper. Before use remove the spirit and polish with a clean, old, much-washed handkerchief.

*Thin blood films*—Select as a spreader a glass slide which has a smooth even end. Clean the patient's skin with a cotton wool swab soaked in spirit. Flame the needle. Prick the finger with a short deep stab take a clean slide, invert it, and bring it just into contact with a small drop of blood about  $\frac{1}{2}$  inch from the end of the slide. Apply the spreader at an angle of  $45^\circ$ —wait until the blood runs by capillary attraction between the spreader and the slide and then draw the film. It is important that the blood should follow the spreader and

that the spreader should not push the blood in front of it. Write the patient's name on the slide with the needle as soon as the film is dry.

*Thick films*—Invert the clean slide. Bring it just to touch the drop of blood issuing from the finger and take up a small drop slightly larger than a pin's head. With a needle spread the drop of blood into an even thick film covering the  $\frac{1}{2}$ -inch square.

The thick film should be stained with Field's stain (1941). Thick and thin smears can be taken on the same slide—the thin film on  $\frac{2}{3}$  of the slide, the thick one at the other end, the slide being divided by a grease pencil line.

*Staining the film*—Leishman's stain is the best known and most useful for thin films. Red blood corpuscles are stained transparent bluish purple, nuclei of leucocytes shades of violet, platelets purple, cytoplasm of malaria parasites bright Cambridge blue and their chromatin red. Fresh distilled water, which is neutral or faintly alkaline (PH 7.2), must be used.

- (1) Lay the slide film side upwards on the staining rack and level.
- (2) Drop the stain from the bottle till whole surface is covered, count 25 slowly.
- (3) Drop on to the slide double the number of drops of pure freshly distilled water, mix intimately, leave for 10 to 15 minutes.
- (4) Fill a bowl with distilled or tap water, take the slide, still covered with the stain, and plunge it into the bowl so that the stain floods off.
- (5) Transfer to a petri-dish full of either distilled or tap water and soak in the dish until film, which is blue green, turns to faint pink; this takes a minute or less.

Remove the slide from the dish and place it against a vertical wall to dry. *Never blot the film*. When finished glance through the film with the  $\frac{1}{6}$  lens before the  $\frac{1}{12}$  to see if the leucocytes are deeply stained. If this is the case the malarial parasites will presumably be well stained.

### MALARIA CONTROL

Each locality has its own special conditions, which call for close study by an expert before embarking on expensive measures of control. Methods found successful in one place fail completely in another owing to different habits of the same anopheline mosquito in different districts.

The closest liaison should be maintained with the local health authorities and, in the case of troops, with the Malaria Field Laboratory.

Measures of control involve the patient with malaria, the susceptible population, and the malaria-carrying mosquito.

### 1 *Control of the Patient with Malaria*

(a) Every patient with malaria should be efficiently treated with atabrin and plasmoquin in order to prevent him becoming a gametocyte carrier

(b) Every patient with malaria should be protected from mosquito attacks by means of mosquito nets or screening

### 2. *Control of the Susceptible Population*

(a) All Europeans living in a highly malarious area, who by reason of their work run frequent risks of infection, should take prophylactic atabrin, or if it is not available quinine

(b) The use of the mosquito net is undoubtedly the most important measure of personal protection against malaria. Rectangular nets are the best bell-shaped nets with a circular top are the next best. The material should be white in order to facilitate the detection of mosquitoes. Nets should be let down before dusk, stretched tight, and searched for mosquitoes before retiring. The bed should be large and the net larger

(c) Screening or mosquito-proofing of all rooms and verandahs with wire gauze is an excellent measure of protection and should invariably be carried out in war time for all hospitals and base units for the benefit of the night staff. Overhead fans in screened rooms are desirable, as otherwise the door is likely to be left open to prevent the room getting too stuffy. Double doors with a vestibule are desirable

(d) The danger period is from sundown to sunrise therefore shorts and rolled up sleeves should be forbidden after sunset. Shorts, if worn in the evening, should have knee-flaps. Gloves, veils and mosquito boots are very useful. If mosquito boots are not available a pillow-case into which one puts one's feet and ties above the knees, is effective. Mosquitoes are most attracted by black or blue they avoid white or yellow

(e) Cuhfuges or mosquito repellants should be used. All essential oils are good, but their effect wears off in a short time. They act by obscuring the attractiveness to the mosquito of the human odour

### 3 *Control of the Malaria-carrying Mosquito (Fig 38)*

#### *A Measures against the adult*

(a) Swatting with a fly swat, especially in cupboards, under the beds, and other dark places in the day time

(b) A general purpose spray is easily obtained with ordinary kerosene, which is improved by the addition of pyrethrum. Carbon

tetrachloride is dangerous and should not be used in a spray For lofty rooms and marquees which cannot be sprayed, Buxton's steam kerosene vaporiser is very efficient

(c) Tangle foot is a mixture of crude castor oil, 5 parts, with

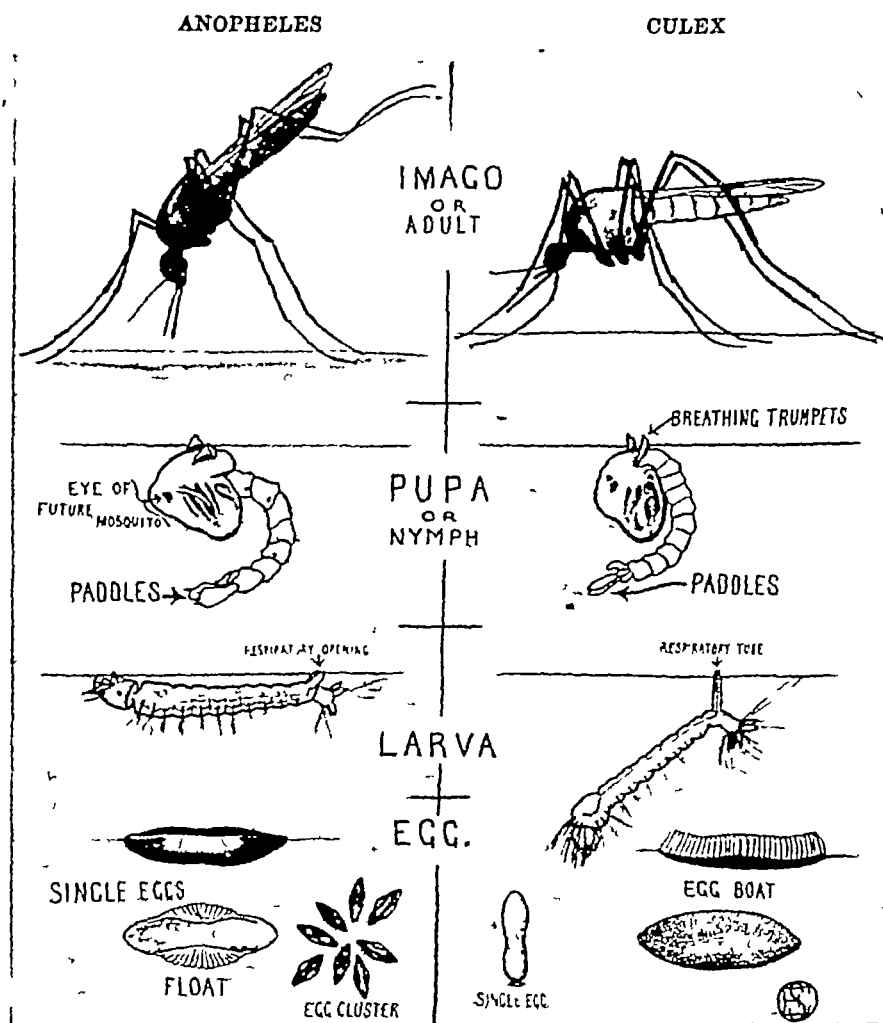


FIG 41 — Comparison between the adult, pupal and larval stages of anopheline and culicine mosquitoes In the adult in the resting position, the body of the former is inclined at a high angle while that of the latter lies parallel to the surface on which it is resting The anopheline wings are usually spotted, the culicine clear The anopheline larva lies flat, at or on the surface, and has no long breathing tube projecting from the last abdominal segment but one The culicine larva hangs down at an angle of  $45^\circ$  with only the tip of its long syphon or breathing tube projecting above the surface The anopheline head is much smaller than the culicine and the body has more dorsal and lateral hairs The anopheline eggs are laid on the surface in star and ribbon patterns, the culicine in compact raft-like masses about one fifth of an inch long

commercial resin, 8 parts, heated and smeared on wires hung from the ceiling

B *Measures against the mosquito in the aquatic stage* (ovum, larva and pupa)

(a) Clearing scrub and jungle

(b) Drainage and filling in surface and subsoil water should be carried out under expert engineering and entomological advice

(c) Bonification, i.e. work carried out to make malarious regions more healthy and suitable for agriculture, thus improving the social and economic life of the inhabitants.

(d) Oiling pools, ponds and slow rivers with crude heavy oil (2 parts) paraffin (1 part) and crude castor oil (2 per cent) kills ova, pupae and larvae.

(e) Spraying pools ponds and slow rivers with Paris green which is a direct chemical poison to larvae only. It should be applied every eight days by a blower. It is used in dilutions of 1 to 5 per cent. with road dust, slaked lime, ashes or sand. It does not kill fish nor render water unsuitable for domestic purposes

(f) Certain species of surface-feeding fish have been found to be useful supplements to basic mosquito control measures. These are the killfishes (*Fundulus*) in salt water and the top minnow or millions (*Gambusia*) in fresh water. Gold fish keep pools and tanks free from culicine mosquitoes, but are not suitable for anophiles control.

For further details the reader is referred to *Malaria Control by Anti-mosquito Measures* by Covell and *Mosquito Control by Harms* and Grey

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## CHAPTER XXIV

### MENINGOCOCCAL FEVER

#### MENINGOCOCCAL SEPTICÆMIA AND CEREBROSPINAL MENINGITIS

BY MAJOR-GENERAL A W STOTT, F R C P

Cerebrospinal fever first assumed importance as one of the "medical diseases of war" in 1914-18. In previous campaigns, except for outbreaks in French garrisons during the Napoleonic Wars and an epidemic in the Army of the Potomac in the American Civil War, the disease had been singularly absent (Foster and Gordon, 1922). The first clear account of epidemic cerebrospinal fever relates to an outbreak which occurred in Geneva in 1805 (Foster and Gaskell, 1916). Since that date many epidemics of varying size, lasting for a few consecutive years and separated by quiescent periods, have been reported from several parts of the world. The British Isles were hardly affected until the early years of this century. In 1906-08 more serious but localised outbreaks occurred in seaports such as Belfast, Glasgow and Edinburgh. The continuance of the disease in small epidemics and sporadically led to its inclusion among the notifiable diseases in 1912. In that and in each of the two succeeding years about 300 cases were notified. In the winter and spring following the outbreak of war in 1914 a much more serious epidemic began. This was not a localised epidemic but widespread over the country, affecting both the civilian population and the troops, especially in centres where the latter were concentrated, and it persisted until the war ended. In the five years 1914-18 10,259 cases were reported with 5,883 deaths, a fatality rate of 57 per cent. During the period 1915-18 there were 1,583 cases in the B E F. in France, but no serious outbreak occurred in any other theatre of war (Foster and Gordon). The epidemic rapidly abated when the war ended, and for some years the number of sporadic cases approximated to the pre-war figures. There was another epidemic in 1931 and 1932 of over 4,000 cases, and since that date the annual notifications have been between 800 and 1,000. With this evidence of a widespread infection and with the experience of the last war it was to be expected that the outbreak of the present war would soon lead to an even

worse epidemic, for the circumstances of modern war greatly favour the spread of the meningococcus. Final figures for the epidemic of 1939-40 are not yet available, but provisional figures for the first quarter of 1940 show that there were 5 093 cases among civilians and troops with 1 120 deaths, a fatality rate of 22 per cent. (Banks, 1940) When to this total there are added the figures for the remaining nine months of the year, it will probably be found that there were in 1940 twice as many cases as in 1915 the worst year of the last war and from the experience then gained we may expect the disease will remain epidemic until the end of the war

### The Natural History of Meningococcal Infection

In the early days of the last war it was generally believed that the meningococcus reached the meninges by direct spread from the nasopharynx (Foster and Gaskell, 1916) By the end of the war however many workers considered it more probable that the meninges were infected from the blood-stream (Foster and Gordon, 1922) This opinion is now almost universally held and it enables us to view in clearer perspective the natural history of meningococcal infection.

The meningococcus gains a lodgment in the nasopharynx, of which it is not a normal inhabitant. Some hold that this constitutes infection, others that the organism is merely 'carried' in healthy tissues. It is certain that the majority of people who thus harbour the organism do not suffer from any easily recognisable symptoms or physical signs of local disease. Murray (1929) found, however clear evidence of inflammation of the nasopharyngeal mucosa in patients dying soon after the onset of cerebro-spinal fever and believed that meningococci entered the blood-stream by way of this damaged tissue. Patients with cerebrospinal fever sometimes remember symptoms suggestive of nasopharyngeal inflammation previous to the sudden onset of meningitis. Brinton (1941) suggests that every carrier and all patients with cerebro-spinal fever suffer from local changes in the nasopharynx identical with, but usually much slighter than, those described by Murray. In the great majority meningococci disappear from the nasopharynx spontaneously within a week or two. In a smaller proportion of cases, especially when there is some local abnormality such as adenoid vegetations, the organism persists for months, and in a still smaller number—those who are "susceptible"—the

organism enters the blood-stream, when, consequent upon unknown factors, one or more of the following events may ensue.

(1) A fulminating septicæmia may develop, which may kill the patient within twelve to thirty-six hours and before the meninges have been affected.

(2) The organism, while persisting for a variable time in the blood-stream, may rapidly pass the "meningeal barrier" and cause a diffuse purulent cerebrospinal meningitis. The patient may recover, or he may die from toxæmia, from the effects of unrelieved intracranial pressure or from some other complication of the disease. It is not certainly known how the organism reaches the meninges, but Brinton (1941) is probably correct in believing that it is by a spread from the multiple metastatic small lesions known to be present within the substance of the central nervous system at an early stage

(3) The organism may cause a milder septicæmia, which may persist for weeks, months or even years. This is the condition known as chronic meningococcal septicæmia. The patient may recover either spontaneously or as the result of treatment, or he may develop meningitis at any period during the course of the illness, or he may die as a result of some other complication of this form of the infection.

The essence of meningococcal infection, therefore, is a septicæmia, and cerebrospinal meningitis, commonly looked upon as constituting the disease, is in reality but a complication, although the most frequent and important one, of this septicæmia.

This wider view of the natural history of meningococcal infection should be reflected in its nomenclature. The use of the name meningococcal fever, on the analogy of streptococcal fever and staphylococcal fever (Ryle, 1930, 1931) would embrace all varieties of meningococcal infection.

### Ætiology.

The *Neisseria meningitidis*, commonly known as the meningococcus, is a gram-negative diplococcus. It requires for its first isolation enriched media such as blood-agar or serum-agar, an incubator temperature between 30° and 40° C, and a plentiful supply of oxygen, although it has recently been shown that growth may be more marked if 10 per cent of carbon dioxide is also present. Meningococci present serological differences, and at present they are divided into Group I organisms (Gordon's Types 1 and 3) and

Group II organisms (Gordon's Types 2 and 4) Meningococci of Group I are the more virulent and prevail in an epidemic while most sporadic cases are caused by Group II organisms.

Although not a normal inhabitant of the nasopharynx, the meningococcus is found in that situation in 3 to 5 per cent. of the community even in non-epidemic periods when but few sporadic cases are occurring. In favourable conditions the number of those harbouring the organism rises considerably so that more than 70 per cent. of a community may be carriers. Such a rise in the carrier rate usually but not invariably, precedes an epidemic.

Of the conditions favouring the rise of the carrier rate the most important is overcrowding, especially in ill ventilated sleeping quarters, and particularly at times when upper respiratory tract catarrh is prevalent for this, with its resulting coughing and sneezing, provides the organism with its usual method of spread—droplet infection. Thus the majority of cases of meningococcal fever occur during the late winter and spring. The disease is rarely spread by case-to-case infection, and more than one case arising in a household is unusual.

Only a very small proportion of carriers develop meningococcal fever. These are the susceptibles which include a very high proportion of infants, children and adolescents.

These aetiological factors explain why meningococcal fever is a "war disease," for in war time the force of circumstances results in the crowding together in ill ventilated sleeping quarters canteens, etc. of large numbers of young troops.

#### ACUTE MENINGOCOCCAL SEPTICÆMIA

This fulminating form occurs in a small percentage of cases of meningococcal fever especially during the earlier stages of an epidemic. Before chemotherapy was available death took place within thirty-six hours and even to-day the fatality rate is high. Death may come before any obvious pathological change in the meninges has occurred, but even after only a few hours of illness Foster and Gaskell (1916) sometimes found an obvious purulent meningitis. At post-mortem examination of five fulminating cases an acute encephalitis was found (Banks, 1940). Sections of the brain tissue showed severe congestion and oedema perivascular infiltration, widespread capillary hæmorrhages and thrombosis, and

extensive nerve-cell degeneration. Meningeal exudate was absent or negligible

The onset is remarkably abrupt and the patient is gravely ill from the very beginning. He may fall down unconscious or be seized with convulsions or violent delirium, or, having gone to bed in his usual health, he may be found unconscious or even dead in his bed in the morning. Soon after the onset large purpuric spots appear in the skin of the trunk, limbs and face. The hæmorrhagic tendency may be further shown by bleeding from the mucous surfaces. Fever is very variable and the temperature may be subnormal. The heart rate is much increased and the pulse weak, even imperceptible. Cyanosis, sweating and dyspnœa with rapid shallow respirations are prominent symptoms, while evidence of meningeal inflammation may be lacking. Unless early and intensive treatment is instituted, the patient will die, the fatal result in young subjects being not infrequently due to vasomotor failure resulting from hæmorrhage into the suprarenal glands—the Waterhouse-Friderichsen syndrome.

### CEREBROSPINAL MENINGITIS

In this form of meningococcal fever some organisms pass, usually soon after they have reached the circulation, from the blood to the meninges, while others persist in the blood-stream for a variable number of days. The effects of the meningeal inflammation usually dominate the clinical picture, but focal encephalitis may occur in addition, and the degree of this, together with the degree of toxæmia resulting from the continuing septicæmia, are probably responsible for the wide variations in severity found in this group. In very acute cases the patient dies within the first five days in a comatose, toxic and dehydrated condition. On the other hand, mild or abortive cases are seen with an illness so slight that its true nature is not at first recognised -

#### Symptoms and Signs

In a majority of cases this form of the disease begins suddenly, often with a rigor, in a patient previously in good health. In a few the onset is more gradual with increasing malaise for a few days. In yet others a carefully taken history shows that they have for some days or weeks been suffering from an unrecognised chronic meningococcal septicæmia before the infection spread to the brain

Headache soon follows the initial rigor and steadily increases, often to an agonising intensity. The pain involves the whole head, though it may be most severe in the frontal or occipital areas. It is never unilateral and is quite unaffected by the usual headache remedies. Relief, sometimes considerable follows the removal of cerebrospinal fluid by lumbar puncture. Accompanying the headache there are often pains in the muscles and joints resembling those of influenza. With increasing headache vomiting usually occurs. It may be repeated frequently for a day or two. Nausea is slight or absent. Soon after the onset the temperature rises, but the degree of fever is very variable and the temperature chart is neither characteristic nor a guide to the severity of the infection. High fever is not common and the temperature usually lies between 100° and 103° F during the acute phase but both mild and severe cases may show little fever. The pulse also has no characteristic features, its frequency is usually increased, sometimes greatly sometimes little in relation to the temperature, but bradycardia is uncommon. Very often within the first twenty four hours muscular rigidity a very important diagnostic sign, is found. This is most easily demonstrated in the muscles at the back of the neck, so that the head cannot be flexed forward towards the chest and attempts to do so cause pain. It is also observed in the hamstring muscles, giving rise to Kernig's sign. When spasm of the neck muscles is severe, retraction of the head results, but this is usually not pronounced and opisthotonos is seldom seen. In order to ease his stiffened muscles the patient often adopts a characteristic attitude in bed—he lies on his side, turned away from the light, with his back and knees flexed and his head retracted.

A rash is present in about half the cases. It usually appears during the first two days. It is hæmorrhagic and occurs in the form of small petechiæ, which do not fade with pressure and are not raised above the skin. The petechiæ vary much in size shape number and shade of redness. The rash rapidly fades within two or three days of its eruption. In severe cases larger purpuric spots are found. Herpes febrilis is common about the mouth and nose but does not appear until the fourth day or later. Erythematous and macular rashes are also described.

All but mild cases show some disorder of the mental functions, although this varies much in degree and kind. There may be drowsiness, restlessness and irritability. In severe cases delirium

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about half the deaths. Other complications occasionally recorded are glycosuria, hyperglycæmic coma and ulceration of purpuric spots. The heart is very rarely affected. Bronchopneumonia is not infrequent in infants and in elderly subjects with a severe infection. As in epidemics of other infectious diseases, mild or abortive cases occur especially when the epidemic is waning, and these are apt to escape recognition and to be diagnosed as influenza. The true nature of this mild febrile illness is shown by the discovery on a careful examination of slight signs of meningeal irritation, such as some stiffness of the neck or hamstring muscles and a cerebrospinal fluid containing an increase of polymorphonuclear cells and protein, though the meningococcus can seldom be found in film or culture. Spontaneous recovery is probably frequent.

With efficient treatment the majority of patients even those with a severe infection, improve so rapidly and the duration of the illness is so shortened that it must be difficult for those now seeing the disease for the first time to recognise the descriptions written during previous epidemics. A few of the acute cases however fail to respond to treatment and die in four or five days from severe dehydration and toxæmia.

#### Diagnosis

Diagnosis at the earliest possible moment after the patient has come under medical care is of the greatest importance for success in treatment, and the suspicion of meningitis should lead to lumbar puncture without delay. If this cannot be carried out at once, the patient should be admitted to hospital as soon as possible after receiving a suitable dose of one of the sulphonamides—details regarding the drug dosage the time of administration and route by which it was given accompanying him.

During the presence of an epidemic the great majority of cases present little difficulty in diagnosis by the end of twenty four hours. Some other illnesses begin suddenly with a rigor quickly followed by malaise headache and vomiting but the presence especially in a young subject, of mental changes a hæmorrhagic rash and muscular stiffness, even slight, in the neck or hamstring muscles should immediately suggest the meningococcus as the probable cause. The diagnosis is made certain by the changes found in the cerebrospinal fluid. The fluid flows out of the needle at an increased pressure it is turbid, often grossly from the great increase of polymorphonuclear cells, of which there may be 20 000

sometimes maniacal, soon gives place to increasing stupor and finally to coma. With the severe grades of clouding of consciousness there is incontinence of urine and faeces. Early retention of urine, without impairment of consciousness, was looked upon as an important and early diagnostic sign in the epidemic of the last war, but accounts of the present epidemic do not mention it, and Brinton (1941) points out that such an occurrence would be difficult to explain on theoretical grounds.

Focal effects of the inflammatory process in the nervous system are not uncommon, nor are they serious with the exception of deafness. Affections of the third, sixth, seventh and eighth cranial nerves lead to diplopia, strabismus, ptosis, facial weakness and deafness. A temporary mono- or hemiplegia sometimes occurs. There are no characteristic changes in the superficial or deep reflexes, nor in the pupils. The onset of deafness always causes anxiety, for although in some cases it is partial and temporary, in perhaps 1 to 3 per cent. of cases it is complete, bilateral and permanent. This is the only serious sequel of meningococcal meningitis today, and as it commonly appears early in the disease it seems probable that chemotherapy will result in an increased incidence because of the higher survival rate.

The continuing septicæmia is responsible for certain metastatic effects which may arise in other parts of the body. In 5 to 10 per cent. of cases there is an arthritis, or more commonly a synovitis, usually confined to one of the larger joints. The joint shows all the evidences of acute inflammation, including an effusion of fluid often considerable in amount. This fluid is turbid and may contain meningococci, but their cultivation from the fluid is unlikely if the patient has been taking a sulphonamide drug. The inflammation soon subsides and the joint is quickly restored to normal function, but in a few cases some stiffness persists for a few weeks. Serious affections of the eye are occasionally seen—iridocyclitis and even a panophthalmitis resulting in destruction of the eyeball. Acute epididymo-orchitis, resolving without any permanent effects, is a less frequent complication than in previous epidemics, for it arose about the end of the first week, by which time the majority of patients have now recovered.

Chemotherapy has also resulted in the virtual disappearance of recrudescence, relapse and the onset of the dreaded complication of hydrocephalus, which in previous epidemics was the cause of

and nodules, varying in size from a few millimetres to a few centimetres in diameter and in colour from pink to dusky red. Some of the papules or nodules may be painful and nearly always some are tender. On pressure the colour fades, but a small central hæmorrhage can often be identified. Petechiæ and large lesions resembling erythema nodosum are also common. Subcutaneous nodules, which may be very tender are sometimes found. The rash may be scanty easily escaping notice, or profuse. It is distributed chiefly on the limbs and on the back of the trunk fewer lesions are found on the chest abdomen and face. Successive crops appear especially with a rise of temperature and each crop lasts for a few days and fades, leaving a slight brownish discoloration.

Fever is either regularly intermittent with a daily rise to 101-105° F or more irregular and relapsing in type short bouts of fever being separated by afebrile periods, during which the patient may feel well and resume his normal life. A rise of temperature may be accompanied by chilly feelings or a rigor a fresh crop of skin lesions and a polymorphonuclear leucocytosis it may be followed by a profuse sweat. The spleen is sometimes palpable.

Cases of chronic meningococcal septicæmia vary much in duration and severity. The illness may be mild, short-lived and end in spontaneous recovery or it may persist for years if unrecognised and untreated. A striking feature, even in these prolonged cases, is the relative absence of debility wasting or anæmia. It is remarkable that complications from bacterial emboli are not more frequent, for it seems probable that many parts of the body must suffer from lesions similar to those found in the skin, from which meningococci have often been recovered. The commonest complication is meningitis, which may arise at any time in the course of the illness. Infective endocarditis and acute epididymitis also occur and nephritis has been described.

Diagnostic proof can be obtained only by the isolation of the meningococcus from the blood-stream. This, however is not an easy matter and blood cultures are often repeatedly negative especially in the early weeks of the infection. Fortunately the disease commonly presents such a distinctive clinical picture that, when once this is known, bedside diagnosis is simple. Rapid cure follows the oral administration of sulphonamides, and treat-

or more per cubic millimetre. A film of the fluid or deposit shows in most cases sufficient Gram-negative diplococci, both intra- and extracellular, to make the diagnosis certain. In about 10 per cent of early cases the fluid may contain no visible micrococci and remain sterile on culture, though showing a purulent reaction. This negative finding strongly suggests that the infection is meningococcal, because in all other forms of purulent meningitis visible bacteria appear in the fluid very early and in considerable numbers. The chemical changes in the fluid—the disappearance of glucose, the increase of protein and the reduction of chlorides—are of less importance in diagnosis.

Other forms of meningitis—tuberculous, pneumococcal, “acute aseptic,” and that resulting from extension of local disease, especially in the ear—sometimes cause difficulty in diagnosis, which is quickly resolved by examination of the cerebrospinal fluid.

Symptoms and signs closely resembling those of meningitis are occasionally seen in acute infections such as pneumonia and influenza. This condition is known as meningism and is also readily distinguished by examination of the cerebrospinal fluid, which shows no abnormality except some increase in pressure.

### CHRONIC MENINGOCOCCAL SEPTICÆMIA

In this interesting form of meningococcal fever the organism persists in the blood-stream for a varying period, even for several years, and causes an illness, usually not severe, with characteristic clinical features. Chronic meningococcal septicæmia was first described by Solomon (1902), and many cases, mostly sporadic, have since been recorded. Rolleston (1919) and Foster and Gordon (1922) recognised its occurrence during the epidemic of the last war, and Stott and Copeman (1941) reported a series of seventeen cases seen in the B.E.F. in France in the early part of 1940, when cerebro-spinal fever was prevalent.

The onset is usually sudden, with malaise, fever and chilly feelings or a rigor. Severe headache and severe migratory pains in muscles and joints are common. Effusion into a large joint is frequent. Within a few days a skin rash appears. Many different lesions have been described and more than one type can often be found in each case. The commonest lesions are macules, papules

has been devised by Banks (1940 1941) and has given excellent results in his hands

I. Initial period	{	mild case	2 days			
		moderate case	2½ days			
		severe case	3 days			
			over			
Age-group		0-2	2-5	5-10	10-15	15
Daily dose (grm.)		3	4½	6	7½	9
4-hourly dose (grm.)		½	½	1	1½	1½
II Middle period	2 days	½ of dose.				
III. Final period	2 days	½ of dose.				
Duration of administration		6 to 7 days. Total amount				
(adults) 37-46 grm.						
Loading dose (1st and 2nd)		add ½ grm. below 5 years add				
½ grm. above 5 years.						
No loading dose should be given to infants under 6 months of age nor to those of the lower ages in each dosage age-group.						

In fulminating and very acute cases, and in others when the drug cannot be given by mouth or tube in order to attain a high concentration of the drug in the blood as rapidly as possible, the first or the first and second doses should be given by intravenous injection of the sodium solution of sulphapyridine in amounts equivalent to 2 grm. (6 c cm.) diluted with three volumes of saline solution or of the sodium solution of sulphathiazole or sulphadiazine in amounts equivalent to 1 or 2 grm. For a young child or infant ½ grm. is sufficient the external jugular vein is a convenient site for the injection in infants. After these initial injections the oral route should always be used when possible if the patient is unable to swallow the drug should be given by nasal tube. Intramuscular injection of these highly alkaline and irritating solutions should not be employed unless there is no alternative. No sulphonamide drug should ever be injected intrathecally

Sulphanilamide is less effective against the meningococcus and the most likely to cause toxic effects. It has the advantage however of seldom causing vomiting, and it has been much used as an alternative to the more effective sulphapyridine, when, as frequently happens the latter has had to be temporarily abandoned on account of this troublesome symptom. Sulphathiazole and sulphadiazine are at least as active against the meningococcus as sulphapyridine. The toxic effects of sulphathiazole are slight. Sulphadiazine appears to be the best drug yet introduced, for with the same dosage there is a higher and more sustained concentration in the blood and toxic

ment should not be delayed by repeated attempts to isolate the meningococcus

Unfamiliarity with this common form of meningococcal fever often leads to such diagnoses as acute or subacute rheumatism, erythema nodosum, or influenza

### Treatment.

The introduction of the sulphonamides constituted a remarkable advance in therapeutics, which is full of promise for the future. Successful results have already been achieved in the treatment of several bacterial diseases, but in none is this success more striking than in meningococcal infections. Group I and Group II organisms, sporadic and epidemic, are alike susceptible to certain of these drugs. Their action is not yet understood, but it is known that they are bacteriostatic and not bactericidal.

Success in the treatment of meningococcal infections by sulphonamides will depend upon the observance of certain rules, with which and with the possible toxic effects of the drugs the medical practitioner should be familiar. That this knowledge is not as widespread as it should be is suggested by the fact already mentioned that the mortality in the epidemic of the spring of 1940 was about 22 per cent., although individual workers have reported series of cases with a mortality of 5 per cent and less.

The drugs of the sulphonamide group which are the most useful are sulphanilamide, sulphapyridine, sulphathiazole and sulphadiazine. Whichever drug is used, the following rules should be observed.

(a) Treatment should commence as soon as possible after the onset of the disease

(b) The drug should be given in adequate amount in regard to both individual doses and total quantity, and the first one or two doses in the course should be the largest.

(c) As the drugs are rapidly excreted, dosage should be frequent in order to maintain a high concentration in the blood and cerebro-spinal fluid. It has been found that this is achieved if the drug is given at regular four-hourly intervals during both day and night. The importance of this rule should be impressed upon the nurses in charge of the patient. The tablets should be crushed and given suspended in water or milk.

The following scheme for oral administration of the drugs

sonal toxic effects, which rapidly disappear on withholding the drug

Before the introduction of the sulphonamides the standard method of treating meningococcal infections was by specific serum, and, if meningitis was present, by repeated lumbar puncture in addition. Serum treatment has now been abandoned, for experience has shown that its use in conjunction with sulphonamides confers no advantage.

Drainage of the spinal theca lowers the concentration of sulphonamide in the cerebrospinal fluid. Lumbar puncture should always be performed at the earliest possible moment for diagnostic purposes, but it should not be repeated unless, as infrequently happens, it is rendered necessary by symptoms of continuing high intracranial pressure

The specific effect of chemotherapy must not lead to the neglect of other important essentials in treatment. Although case-to-case infection is rare, the patient should be isolated whenever possible and be nursed with the precautions usual in infectious disease. Good nursing is essential especially in the case of comatose and delirious patients. An adequate supply of fluids—at least four pints daily for an adult—is of the greatest importance in every case in order to prevent not only dehydration and its serious consequences but also the urinary complications already mentioned. If the patient is unable to swallow fluid containing a liberal amount of glucose and the sulphonamide should be administered regularly by tube. The rectal and intravenous routes are also available in case of need.

An enema should be given on alternate days if the bowel fails to act naturally and retention of urine necessitates the use of a catheter

Severe headache and restlessness are usually relieved by lumbar puncture, but sedatives such as paraldehyde given rectally or a hypodermic injection of morphia are sometimes needed. For the acute adrenal insufficiency which results from adrenal hæmorrhage and is very fatal in fulminating cases, Hughes (1940) advises large doses of sodium chloride and also adrenal cortical hormone. Recovery has been recorded.

#### Course and Prognosis

The sulphonamides in adequate dosage have not only considerably reduced the mortality of meningococcal infections. They have in addition lessened their severity shortened the course and con

effects are said to be rare. Unfortunately these two drugs are not yet available in sufficient quantities to allow of their general use.

The dosage recommended by Banks is high, especially in the lower age groups, but he believes that lower dosages are dangerous. There is evidence that initial low dosage causes the condition known as drug-fastness, so that the organisms are resistant to subsequent higher doses. Infants and children tolerate high doses remarkably well, and at any age serious toxic effects are rare if the drug is not administered for a longer period than six or seven days. In chronic meningococcal septicæmia lower doses are effective. An initial oral dose of 2 grm should be followed by four-hourly doses of 1 grm until the temperature falls to normal. Then 1 grm should be given three times daily for another four to six days.

Sulphanilamide nearly always causes cyanosis, usually from the formation of methæmoglobin, but it has little importance during the treatment of meningococcal infections. The troublesome nausea and vomiting of sulphapyridine have already been mentioned. All these drugs not infrequently cause "drug fever" and rashes, generally morbilliform, which usually appear between the seventh and tenth day of administration and so should not often occur during the treatment of meningococcal infections. Brinton points out that their appearance is a useful warning that more serious toxic effects may follow, unless administration of the drug is stopped at once. He advises an immediate blood count if fever or rash appears, so that early granulocytopenia may be recognised. This is the most serious toxic effect of sulphonamide drugs and is fortunately very rare. Excessive or prolonged dosage is not always responsible and idiosyncrasy probably always plays a part. Pent-nucleotide—10 c c by intramuscular injection six-hourly until the blood shows a return to normal—is the only effective treatment.

Hæmaturia, oliguria and even anuria may occur during the administration of sulphonamide drugs, especially sulphapyridine. These symptoms result from the drug crystallising out of an acid and concentrated urine in the renal tubules, pelvis or ureters. The sharp crystals cause hæmaturia by trauma, large collections of crystals may block the outflow of urine and lead to anuria. The drug should be immediately withdrawn and the patient should be given large amounts of alkaline fluid. Measurement of the daily output of urine is important.

Severe mental changes, such as confusion and delusions, are occa-

Medical officers of units can do a great deal by giving simple talks to officers and men explaining the possible gravity of the disease, its causes, mode of transmission and the methods of prevention.

When seeking a carrier care must be taken in obtaining a nasopharyngeal culture that the swab does not become contaminated by saprophytes in the mouth. A long swab of the West type guarded within a glass tube is the best its tip remaining within the tube until both have passed the anterior faucial pillars, when it is thrust out to collect mucus from the nasopharynx and pulled back within the tube before withdrawal through the mouth. The swab must then be kept warm and moist during transit to the laboratory as the meningococcus is very readily killed by cold and drying. Should a carrier be discovered, it has been found that 1 grm. of sulphanilamide three times a day for six days will usually cause the disappearance of the meningococcus, but it is not known how long this freedom lasts.

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valescence, and greatly eased the medical and nursing care. Improvement in the patient's condition is often obvious within twenty-four hours, even in very severe infections, and the majority of patients are well by the end of a week and are fit to leave hospital in another week or two. Convalescence also is usually rapid and undisturbed, and most patients are fit to resume a normal life in six to eight weeks after the onset of the illness. In some patients general nervous symptoms, such as headache, giddiness, slight emotional disturbances and insomnia, make their appearance when normal activities are resumed. As with the similar symptoms which often follow head injuries, psychogenic factors are often important, especially in patients with a history of previous nervous instability. Recovery is usually complete and is hastened by treatment similar to that described for the functional sequels of head injuries.

### Prevention and Control.

The control of meningococcal infections presents a very difficult problem, especially in modern warfare. Nevertheless much can be achieved by counteracting as far as possible the conditions which are known to favour the spread of the meningococcus.

Cases of the disease should be notified to the health authorities without delay, the patient's clothing, bedding and feeding utensils should be disinfected, and his room thoroughly scrubbed and aired.

During the last war there was an intensive search for carriers among contacts and others, which entailed a great deal of laborious work. Carriers were segregated and given local treatment to the throat, but these methods failed to influence the spread of the infection and they have now been abandoned, even in the case of immediate contacts.

The most important precaution is the prevention of overcrowding, especially in sleeping quarters. There should be at least six feet between the centre lines of adjoining beds, and, if this is impossible, every alternate bed should be turned so that its occupant lies with his head opposite his neighbour's feet. In no case should a distance of less than five feet be allowed. In some buildings it may be possible to gain extra space by placing beds in the middle of the room. All rooms should have doors and windows widely open during the day to allow free access of sunlight and air, and every effort should be made to provide some kind of light-screened ventilation during the night.

Cappadocia by Aretaeus in the first century A.D. and Trousseau points out that under the names Egyptian and Syriac ulcer the disease has been endemic in those countries from time immemorial. He himself employs the synonym *mal égyptique* in his clinical lectures delivered in the sixties of the last century. In Egypt diphtheria is, or was until recently when mass immunisation of children was introduced, the most serious of the endemic diseases, attended by a very high case fatality rate. Although a common disease in Egypt, there is general agreement that in the tropical regions of Africa diphtheria occurs only sporadically. In Tanganyika, Schick tests and antitoxin estimations showed that the natives possessed a high degree of inherent immunity (Kleine and Kröo 1930). In Palestine Mann and Khgler (1929) have shown that among the native population the disease occurs in so mild a form as often to pass unrecognised, and this is also true of Syria (Parr Goodale and Krischner, 1930).

Although largely independent of climate, prevalence of diphtheria in endemic areas is influenced by weather. In countries of temperate climate the disease is most prevalent in the autumn and winter months in common with other acute infections of the upper respiratory tract. Rogers (1919) says that most cases occur in Calcutta in the "cold weather" season but that they are very rare during the rains. In Indian hill-stations diphtheria may be encountered at any season. The disease among the native populations tends to be mild and clinically unrecognisable. Localised outbreaks among regular British troops and their families are not rare and some of these have been traced to native servants who were carriers.\* Carriers and those suffering from abortive clinical attacks the *formes frustes* maintain the chain of infection chiefly by droplet spray or droplet nuclei but fomites such as cups and spoons and infected dust also play a part. The importance of overcrowding, especially in sleeping quarters in barracks, in the dissemination of infection is well recognised. As Glover (1918) showed of the spread of the meningococcus in barracks and Dudley (1923) at the Royal Naval School at Greenwich similarly of the diphtheria bacillus and the haemolytic streptococcus, the most important factor is the distance between beds. Gordon Cheyne (1942) says that in the

\* For a comprehensive survey with many references, of diphtheria prevalence in non-European countries, see J. Graham Forbes (1932), *Bull. Hyg.* 7 737

## CHAPTER XXV

### DIPHTHERIA

By E H R. HARRIES, M D , F R C P.

In the Official History of the War of 1914-18 it is recorded that "diphtheria never made much headway among British troops in France, although there was a tendency to increase in 1917, when the highest monthly rate reached 13·6 per 100,000." It is stated, however, that the disease was very prevalent among the troops in Sinai and Palestine. In Mesopotamia (Iraq) the official notifications appear to have been few, but my personal experience in one of the isolation hospitals in that theatre of war goes to show that, for one reason or another, the actual cases exceeded the notifications. To a much greater extent than in civil life non-respiratory forms of diphtheria occur among troops on active service, either from the infection of wounds—a source of trouble in German military hospitals in the last war—or, especially in the Near and Middle East, from the secondary infection of skin lesions.

Since the last war diphtheria has been added to the list of preventable diseases, and Canadian and American troops drawn from the large cities must include a high proportion artificially immunised in childhood. In Great Britain, however, until very recently mass immunisation upon an adequate scale has not been carried out except in a very few centres. It is therefore safe to say that the number of British recruits immunised in their pre-school or school years form an inconsiderable proportion of the whole, so inconsiderable that unless augmented after entry to the Services no appreciable effect upon incidence is expected.

In the following pages no attempt has been made to present a complete account of diphtheria—only those aspects which may concern medical officers in the Forces on active service are discussed.

**Aetiology.**

Diphtheria occurs in nearly all parts of the world, it is singularly independent of climate. The common belief that sub-tropical and tropical countries are exempt and that the disease is the perquisite of populations living in the cities of countries of temperate climate is fallacious. Indeed the first recognisable account was written in

strains differ somewhat in morphology and cultural reactions. *Gravis* is short, stains uniformly and shows few granules *intermedius* shows bands or bars, but if grown in tryptic agar no granules. All three strains ferment glucose but not saccharose *gravis* strains in addition ferment starch and glycogen. *Mitis* strains alone are hæmolytic.

The accurate and relatively speedy recognition of the diphtheria bacillus has been much facilitated by the use of tellurite media, of which there are several variants, e.g. the 'chocolate' (tellurite and laked blood) medium of Neill (1937) and the tellurite blood-agar of Hoyle (1941). Tellurite media now used for routine investigation in most laboratories owing to their selective character secure a higher percentage of positive results than the old Löffler serum (99·6 per cent. 81·5 per cent. (Hoyle)) and also because the distinctive colony morphology of the three strains permits in experienced hands of their identification by means of a hand lens. The appearance and size of the colonies differs somewhat with the medium used but in general *gravis* colonies appear as grey rosettes or marguerite daisies *intermedius* colonies are flat and present a grey halo around a black centre and *mitis* colonies are black, convex, smooth and shining. Since practically all *gravis* and *intermedius* strains are virulent it is only in the case of *mitis* strains that guinea pig virulence tests may be called for. Hynes (1942) concludes that in London Neill's is superior to Hoyle's medium, particularly for detecting convalescent carriers or patients whose swabs give a rather poor growth of *O. diphtheriæ*. With either medium a positive result can be confidently reported after eighteen hours' incubation, but a negative result should not be reported until it has been confirmed by a further day's incubation. Hynes considers that differentiation by colony morphology and the distinction of *O. diphtheriæ* from other organisms is better with Neill's medium, but is adequate with Hoyle's.

#### Pathogenesis

The main points in the pathogenesis of diphtheria may be recounted: the formation at the site of infection of a more or less typical pseudo-membrane; the production at and dissemination from the site to all the tissues of the body of an exotoxin possessing special affinity for cardiac and nervous structures; the rapid and irreversible fixation of this toxin in the tissues with clinical results which depend upon the amount of toxin so fixed. The amount dis-

Army an endeavour is always made to separate heads and that double bunking of barracks has been of great value

The results of infection clearly depend upon the state of immunity of those infected and upon the dosage and virulence of the strain of the *Corynebacterium diphtheriæ*. In civil life in England the great majority of cases of clinical diphtheria and nearly all the fatal ones occur under the age of fifteen years. In a community of adults the percentage of natural immunes may range from 50 to 70 per cent or more depending upon the environment during childhood. The higher percentages are found among the town-bred, the lower among those brought up in country districts, where the opportunities for acquiring immunity by the aggregation of small specific stimuli are fewer. Some so immunised remain carriers. If necessary or feasible, the incidence of immunes is ascertainable by Schick-testing and may of course be increased by artificial immunisation. An epidemic of diphtheria is preceded and accompanied by a rise in the carrier rate (Dudley), similar to that shown to occur in the case of cerebrospinal fever by Glover. During the epidemic of diphtheria there is a "coincident epidemic of immunisation" (Dudley)

### Bacteriology.

The morphology of the *Corynebacterium diphtheriæ* (Klebs-Löffler bacillus) is well shown in a smear stained with Albert's stain\*. Examination shows the criss-cross arrangement of the bacilli, their bodies stained green, the bands or bars dark green, and the metachromatic granules (polar bodies) nearly black. It must be emphasised that morphological examination of a stained smear is insufficient for accurate diagnosis of the diphtheria bacillus. Although valuable support may be afforded to clinical diagnosis of a suspicious case of faucial diphtheria, no reliance can be placed upon smears in the detection of carriers. Morphological appearances are deceptive except to the skilled bacteriologist: the organism may be a harmless diphtheroid or a true diphtheria bacillus of an avirulent strain.

There are three strains, *gravis*, *intermedius* and *mitis*, of *C. diphtheriæ*. Although all produce the same toxin, the first two are commonly, but not always, associated with severe clinical attacks, the third with less toxæmia and with laryngeal diphtheria. The three

\* Toluidin blue and methyl or malachite green, counterstained with Gram's iodine solution.

*gravis* and *intermedius* strains are more rapidly invasive than *mitis* and that within twenty four hours of infection the patient may be seriously ill, but I am sceptical about incubation periods of as long as a week unless the anterior nasal form is in question. In this form, as in primary non respiratory diphtheria the length of incubation must usually be a matter for surmise since the occasion of infection is usually unknown and invasion clinically inappreciable.

(i) *Anterior nasal diphtheria* is characterised typically by a yellowish blood-stained discharge which may be unilateral or bilateral. Membranes may or may not be visible within the nares the site may be too far back on the septum or turbinates or none may be produced, as in the atypical catarrhal form which can only be recognised bacteriologically. Nasal diphtheria is associated as a rule with excoriation of the skin beneath the nostrils and sometimes by superficial cutaneous plaques upon the face, the latter caused by auto-inoculation. Departure from health rarely amounts to more than debility thus the patient is ambulant until the condition is detected. This fact and the overt character of the focus render the subject of anterior nasal diphtheria a far more dangerous source of infection than the occult carrier. The condition is uncommon in adults. The rhinitis caused by a foreign body may simulate nasal diphtheria or the two conditions may be combined.

(ii) *Faucial (tonsillar) diphtheria* has usually a quiet rarely abrupt, onset. There may be little or no complaint of sore throat, and it is a wise rule always first to examine the throat of any patient who complains of headache lassitude, anorexia and possibly nausea in order to exclude diphtheria. The face is pale rarely flushed, and wears an expression of fatigue. Pyrexia, if it occurs, is slight or moderate. The pulse is rapid, small and easily compressible. Albuminuria is usual in the more severe attacks. The faucial mucosa may be merely injected and no membrane may be visible then or later. Such cases of catarrhal diphtheria, only to be confirmed bacteriologically are particularly liable to occur among adults possessing considerable basal immunity.

As a rule, however if no membrane is to be seen at the first examination it will form during the ensuing twelve hours and thereafter unless checked by antitoxin, is likely to spread from tonsils to fauces and ultimately perhaps involve adjacent pharyngeal mucosa (pharyngeal diphtheria).

Typical diphtheria membrane consists of a raised patch so closely

seminated may be so small or the patient's basal antitoxic immunity so high or so speedily augmented by therapeutic antitoxin that the clinical effects of damage by toxin are negligible. On the other hand, the amount disseminated may be so great or the patient's basal immunity so slight or so tardily augmented that the myocardium is overwhelmed within the first week of the disease and the patient dies in the acute toxæmic phase. Or the damage wrought during this phase of toxæmia may become clinically manifest only after a lag period and then in the form of "late" circulatory impairment or failure, central or peripheral or both, or of characteristic palsies of central and later still of peripheral origin. The patient may run the whole gamut of these post-toxæmic happenings and yet recover completely from his disease. But having recovered he may still remain for a variable period a convalescent carrier.

It is important to add that diphtheritic toxæmia, unless slight in degree, causes a loss of glycogen from the liver and the bundle of His, a preliminary hyperglycæmia is followed by transient hypoglycæmia and this in turn by hyperglycæmia again. It is believed that the production of insulin is inhibited (Schwentker and Noel, 1929). The blood-sugar curve shows a lag of diabetic type, the degree of lag being proportional to the toxæmia (Benn, Hughes and Alstead, 1932). These facts constitute the rationale of accessory glucose therapy.

The essential cardiac lesion is a toxic parenchymatous hyaline degeneration, often combined with fatty degenerative infiltration (Warthin, 1924). The bundle of His may be completely disorganised. Except in the rare hemiplegia due usually to embolism of the middle cerebral artery, there are no naked eye changes of nervous tissues, but histologically degenerative processes are seen in the medullary nuclei, the spinal cord and peripheral nerves. Changes also occur in the suprarenal glands, liver, kidneys, lymphatic glands and the reticulo-endothelial system.

### Symptoms.

The focus of infection is usually faucial, but extra-faucial foci are not uncommon. Combinations of the two forms are by no means rare, but in such cases the extra-faucial focus is usually secondary and may be initiated by auto-inoculation.

The *incubation period* of respiratory diphtheria is not well defined, but commonly ranges between two and four days with limits of one to six or seven days. There is, I think, no doubt that

the *O. diphtheriae* during an attack of diphtheria of an existing ear discharge is common. But it is to be borne in mind that purulent ear-discharges frequently contain diphtheroids, and therefore accurate bacteriological identification is essential before concluding that the patient is an aural carrier. Diphtheria of the external ear may affect the meatus, membrane being visible, or the pinna, in the latter it takes the form of an intractable eczematous patch.

(v) *Conjunctival diphtheria* is usually secondary to an anterior nasal focus, infection spreading by way of the naso-lachrymal duct. The condition may be confined to the palpebral conjunctiva, but if membrane is extensive or treatment is delayed the ocular conjunctiva is soon involved as well. Slight infections of the palpebral conjunctiva result in small patches of membrane visible on eversion of the lids. Severe or neglected infections result in oedema of the lids and a muco-purulent discharge. Eversion of the lids becomes most difficult, but if accomplished the conjunctival surfaces are seen to be covered with thick membrane. By this time too the ocular conjunctiva has almost certainly been invaded, and unless treatment is prompt and adequate the globe rapidly becomes disorganised and may require excision to prevent sympathetic panophthalmitis. Although ophthalmic advice should if available always be sought, it is well meanwhile not to delay antitoxin treatment, and in addition to dilate the pupil with atropine and to irrigate with boracic lotion or some other non irritating antiseptic. In spite of the fact that a diagnosis of diphtheritic infection of the conjunctiva is not uncommonly mistaken, specific and adjuvant treatment should never wait for bacteriological confirmation.

(vi) *Diphtheria of cutaneous lesions*—On cutaneous surfaces the diphtheria bacillus, like the hæmolytic streptococcus in erysipelas grows only at the site of an abrasion puncture or other breach of skin surface, or as a secondary invader of a pyococcal skin lesion, particularly if this be vesicular or bullous in character. The bacillus does not invade intact cutaneous tissue. Examples of cutaneous diphtheria have already been mentioned—the facial plaques associated with anterior nasal diphtheria and the intractable eczematous patches which may occur upon the pinna. To these may be added the sub-acute type of *varicella gangrenosa* in which the lesions become secondarily infected with the diphtheria bacillus by auto-inoculation or otherwise. The occurrence of diphtheritic

applied to the stratified epithelium of the tonsil that when removed, as it may be with some difficulty by a firm scraping movement with the tip of the spatula, it leaves a bleeding or oozing surface. The upper surface of the detached fragment is smooth but the lower presents a shaggy blood-flecked appearance, best seen when the fragment is placed in a vessel of water, to the bottom of which it sinks intact—a rough but valuable confirmatory procedure. The colour, thickness and extent of membrane vary. Recent membrane is white or creamy-white, darkening later to yellow, brown and ultimately brownish-black. Recent membrane is thin with an active thinner spreading edge. Older membrane is thicker and the edge definite and possibly heaped-up. It is important to note that membrane formed as the result of a *gravis*-strain infection tends not only to spread rapidly in a thin layer but to present a characteristic glairy appearance in its earlier stages. In milder grades of diphtheria commonly no zone of hyperæmia surrounds the patch of membrane, but in more severe infection such a zone is present and in the most severe local œdema of the mucosa as well. Characteristic fœtor is usual in severe cases. Of serious import as an index of a considerable or dangerous output of toxin is the enlargement of the relative lymphatic glands and the associated œdema of periglandular tissue—the extreme degree of which is known as “bull-neck.” In cases of this degree of severity, the so-called toxic or hypertoxic diphtheria, the post-nasal space is nearly always involved, with the result that muco-pus may be seen trickling down the posterior pharyngeal wall and escaping from the nostrils (c.f. anterior nasal diphtheria).

In toxic diphtheria the patient is pallid, inert and apathetic, and the pulse is rapid, possibly irregular and of very poor quality. Nausea and vomiting and oliguria with albuminuria are usual. In hypertoxic diphtheria these signs and symptoms are enhanced in degree, and to them may be added petechiæ into the skin and, of gravest prognosis, hæmorrhages from mucous membranes.

(iii) *Laryngeal diphtheria* in the adult, as in the child, is in most cases secondary to an extra-laryngeal focus in the upper respiratory tract. The condition is rare in adults and still more rarely necessitates operative interference for the relief of obstruction.

(iv) *Aural diphtheria* is nearly always secondary to a post-nasal infection, spread taking place along the Eustachian tube. Very rarely indeed is the mastoid antrum invaded. Super-infection by

the *C. diphtheriae* during an attack of diphtheria of an existing ear discharge is common. But it is to be borne in mind that purulent ear-discharges frequently contain diphtheroids and therefore accurate bacteriological identification is essential before concluding that the patient is an aural carrier. Diphtheria of the external ear may affect the meatus membrane being visible, or the pinna, in the latter it takes the form of an intractable eczematous patch.

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*varicella gangrenosa* is of particular interest, because the clinical manifestations conform in several respects with a form of cutaneous diphtheria, "desert sore," presently to be described. Owing to the fact that the infected lesions in *v. gangrenosa* are multiple and not synchronous, toxæmia tends in the unrecognised case to be considerable, and indeed the patient may die of myocardial failure, the result of cumulative additions of toxin to the circulation. Short of this the actual cause of the intractable punched-out lesions covered by black sloughs may be suspected only when the patient develops palsies of post-diphtheritic type, and only confirmed by the result of bacteriological examination of the offensive sanious discharge which oozes between the indurated edges of the ulcer and its slough. For it is important to note that in all types of cutaneous and wound diphtheria the formation of the typical pseudo-membrane seen in faucial diphtheria is exceptional.

In the Middle East during the war of 1914-18 the condition variously known as *desert sore*, *veldt sore* or *Barco rot* was of frequent occurrence, and C J Martin (1917) suggested the possibility that the diphtheria bacillus might play a part in its ætiology. Under the name "frontier sore" its occurrence on the North-West Frontier in 1935 was described by Bensted (1936). Cameron and Muir (1942) have reported an outbreak in Northern Palestine and Williams (1943) among troops in England. It has been established by several observers that some desert sores do become secondarily infected with the diphtheria bacillus, particularly when faucial diphtheria is rife among the troops or the inhabitants, and that, as first shown by Walshe (1918), the occurrence of a sore so infected may be followed by typical post-diphtheritic palsies, possibly after the sore itself has healed. Walshe records that in the summer of 1917 in Egypt "diphtheroid rods" were found in septic sores in an area of operations where diphtheria was prevalent. Thereafter numbers of cases of wounds and septic sores followed by polyneuritis were observed, but, the lesions being healed, no bacteriological confirmation was possible. Walshe had little doubt that all were of diphtheritic origin, they were associated in point of time and place with the occurrence of faucial diphtheria and post-diphtheritic paralyses and with the isolation of diphtheroid organisms from the particular type of skin lesion which these cases showed, and, finally, in several instances ciliary paralysis, highly characteristic of diphtheria, occurred. In some cases faucial diph-

thema and septic sores were co-existent and it was difficult to decide upon the primary lesion.

Desert sores, of which Strong (1942) describes two types, occur most frequently upon the dorsum of the foot, the shins and around the external malleolus, less commonly they occur upon the dorsum of the hand and the back of the wrist and they are occasionally seen upon the face. The first type is a chronic ulcer which slowly develops from a painless swelling ("gummatous"). It is surrounded by a circumscribed, reddened glazed area of skin. Two to three weeks later the swelling softens and serous exudation from the summit occurs. This is followed by ulceration and frequently by the formation of a membrane-like deposit resulting ultimately in a punched-out ulcer with indurated margins. There appears to be no impairment of health with this type of ulcer. The second type occurs in cachectic persons and in the early stages has been compared to an area which has been excoriated and inoculated with vaccine virus. This area of intense erythema within a few hours may be surrounded by a circle of vesicles and this again by an inflammatory areola. Subjective pain and tenderness are severe. The area within the ring of vesicles is converted into a dark grey to black pultaceous diphtheritic membrane which when detached reveals fungating granulations covered with greenish yellow pus. Strong's description of this type of ulcer bears a close resemblance to the lesions seen in *caricella gangrenosa* the subject of which usually becomes cachectic. Extensions of area and recrudescences may occur for months. Since diagnosis in the case of healed lesions may of necessity be retrospective Walsh's description of two types of scar may afford valuable assistance. He describes the scar of a small sore as a round reddish purple area, in which the texture of the skin may be normal or in which there may be a sharply circumscribed area of shiny atrophic skin. If unaccompanied by neuritis there is no sensory defect, but if so accompanied some patients showed a definite impairment of sensory acuity to touch and pin prick. The scar of a larger sore manifestly the sequel to Strong's second type of ulcer shows a sharply circumscribed area of pale, shiny hairless skin, pink when recently healed, over which are dotted small, irregular raised nœvoid patches. This area is surrounded by a zone of brownish pigmentation on skin of normal texture with a sharp inner margin. In these cases there is always profound, sometimes complete sensory loss and in most

cases a zone of graded sensory change. Of 30 cases analysed by Walshe palsies followed a single infective focus in 12, while in 18 there were multiple skin lesions

(vii) *Diphtheria of wounds*—There are numerous accounts in the French and German literature of wound diphtheria in military hospitals in the last war, but it is open to question whether the organism implicated was always a true diphtheria bacillus or merely a concomitant diphtheroid. In an intensive study of the flora of military and air-raid wounds during the present war Miles and his co-workers (1940) showed that of 154 swabbings from wounds, taken on admission of soldiers and civilians to advance base hospitals in a London Sector, 10.8 per cent. showed diphtheroids—not further differentiated since the main quest was the hæmolytic streptococcus. Of 105 “Dunkirk wounds” 7.6 per cent. showed diphtheroids, and diphtheroids were isolated from 18.4 per cent. of 49 air-raid cases. The same observers show the trend of the bacterial flora of wounds treated in hospital wards at various periods between wounding and swabbing. Thus diphtheroids were isolated, but not estimated quantitatively, in 15.3 per cent., 11.1 per cent. and 33.4 per cent. when the lapses between wounding and swabbing were one to three, four to twelve and thirteen to forty days respectively. It is not stated whether these diphtheroid organisms included any true diphtheria bacilli.

Wieting (1920), describing wound-diphtheria in Germany in the last war, went so far as to identify the condition with “hospital gangrene,” which at least is grim testimony to the conditions under which the patients were nursed. In his experience empyema wounds were particularly likely sites of infection with the diphtheria bacillus.

In the early stages of infection with the diphtheria bacillus the wound becomes painful and reddened. There is at first an irritating serous discharge, which is replaced in a few hours by a more or less typical pseudo-membrane, grey or greyish yellow in colour, thin or thick. If the membrane is removed it re-forms. The edges of the wound may either become livid, infiltrated and definitely raised (phlegmonous type), or undermined with foetid pus, in which case they are not invariably reddened (ulcerative type). Vesicles may form on the edges of the wound, these may rupture and then become covered with pseudo-membrane. Anschutz and Kisskalt (1919) have described wound diphtheria, in which the diagnosis was made retrospectively upon the occurrence of palsies.

## Complications

Owing to the considerable basal immunity possessed by many adults an attack of diphtheria may be so mild as to pass unrecognised and uncomplicated and may provide only an addition to the dangerous class of carriers. Or the initial focus may escape detection and therefore appropriate treatment a retrospective diagnosis being possible only when the man reports symptoms characteristic of the post-diphtheritic complications—circulatory impairment or palsies or a combination of both.

*Circulatory impairment and failure.*—If other causes of dyspnoea faintness and tachycardia on exertion can be excluded post-diphtheritic myocarditis should always be suspected. Although not at this stage a very hopeful quest, bacteriological confirmation should always be sought swabbings from the fauces or an unhealed wound or sore may still reveal the diphtheria bacillus. Circulatory impairment occurring in the patient already warded and under treatment varies greatly in severity. Nothing may be detectable upon ordinary clinical examination but electrocardiograms if obtainable, may reveal a slurring of the QRS complex with or without aberrations of T in significant leads. The process may not advance further but on the contrary quickly regress. Arrhythmias, such as extra-systoles although not in themselves of much importance, should, like the minor electrocardiographic changes be regarded with caution as possible precursors of more serious trouble. Of greater significance is a weakening of the first sound which approximates in quality to the second sound. Reduplication of the second sound and especially triple rhythm are of serious import.

Circulatory failure as distinct from impairment may be early or late.

*Early circulatory failure* occurs during the first week or ten days of the attack. It is due to acute toxic myocarditis and is part of the syndrome of toxæmia. The toxæmia of diphtheria is manifested by lassitude, apathy and facial pallor succeeded, if the process be not stayed, by prostration, lethargy and greyness with some cyanosis of the face. The intellect may remain clear up to the moment of death or the patient may sink into coma. Occasionally there is terminal muttering delirium. As the condition advances, the rapid, easily compressible pulse becomes irregular and the extremities cold. The temperature drops to subnormal. The area of cardiac dulness is not as a rule increased to percussion. The first sound at

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the apex is soft and impure and there may be a loud systolic murmur localised to the left sternal border, the second sound is accentuated. The liver is enlarged and tender.

*Late circulatory failure* may occur at any time from the end of the second week onwards and may be preceded or accompanied by one or more of the palsies discussed below, if these are multiple the event may be apprehended, but there may be no warning. The onset is sudden. The patient vomits, and the temperature sinks to subnormal. He complains of epicardial pain, which may be anginal in intensity. The general appearance and demeanour rapidly approximate to those noted in early failure. The cardiac signs are more variable. There may be tachycardia or bradycardia. The apex beat is displaced to the left. The first sound may be replaced by a loud localised murmur, the second reduplicated. There may be clinical evidence of complete heart block, but since this in diphtheria is compatible with a high ventricular rate, complete block, like bundle branch block, which is not unusual, may be detected only in an electrocardiogram. Oliguria with albuminuria is usual, anuria is of the worst import. The prognosis of early or late cardiac failure is grave in the extreme, but not hopeless. The intravenous injection of glucose is occasionally followed by dramatic improvement.

*Paralyses* — Walshe (1935) maintains that post-diphtheritic paralysis has three components, analogous with the three components of tetanus, the local (palatal) palsy, the specific accommodation palsy, and the general (polyneuritis) palsy. The first two are central in origin and due to lesions of the central nervous system, whereas the third is probably the result of a blood-borne intoxication and due to a lesion of the peripheral nerve fibres. Walshe (1918) believes that palatal paralysis does not occur except after faucial diphtheria, that polyneuritis follows both faucial and extra-faucial diphtheria equally and irrespective of the site of the infective focus, and that paralysis of accommodation also follows both forms of infection. In extra-faucial diphtheria, as in tetanus, the local paralysis varies with the site of the infective focus. These views have found wide acceptance, and as they are based upon observations of cases occurring among the troops in Egypt during the last war they are of special significance at the present time.

Palatal paralysis may occur as early as the end of the first week or be delayed until the fourth or fifth week of the attack. Its

duration varies from two or three weeks up to two or three months. The nasal voice, limitation of movement on phonation and anaesthesia of the palate typical of the condition in children occur but in adults regurgitation of fluids through the nose is uncommon.

Late palatal paralysis may be combined or followed by pharyngeal paralysis, characterised by dysphagia, the dribbling of mucus from the mouth and a reflex cough caused by mucus entering the respiratory tract the result of which may be fatal aspiration bronchopneumonia. Nasal feeding may become necessary to maintain nutrition. If available an electric suction pump to which is attached a rubber tube, affords great relief the tube is passed through the mouth as far as the pharynx at intervals of two or three hours and the mucus aspirated.

*Paralysis of accommodation* (ciliary paralysis) occurs or at least is detected, somewhat later than palatal paralysis. It is usually purely subjective, the reaction to accommodation being positive. Walshe noted that his patients could read only for two or three minutes before difficulty was noticed. Commonly the patient complains that he can read only large print. To allow the patient to read too early or too long in convalescence is to invite paralysis of accommodation. The duration rarely exceeds two or three weeks.

Strabismus is not uncommon. Ptosis is rare. Paralysis of the lower division of the facial nerve is an occasional concomitant. Aphonia and a paralytic cough indicate laryngeal paralysis. Late to appear and fortunately rare are the two lethal palsies of central, probably bulbar origin *diaphragmatic paralysis* indicated by an immobile or sagging upper abdominal wall and overaction of the intercostal muscles, and *intercostal paralysis* in which the thorax is immobile and the diaphragm overacts. Unless very partial, the prognosis in both these types is bad. It has been improved by the early use of a "breathing machine" (Drinker Both or Paul Bragg curass). So treated, diaphragmatic paralysis may persist for a fortnight and then disappear but not uncommonly in diphtheria the patient is cured of his respiratory paralysis only to succumb to aspiration bronchopneumonia the result of an associated pharyngeal paralysis, or to myocardial failure.

*Peripheral neuritis* occurs late in the attack or may be the first event which leads to a retrospective diagnosis of diphtheria. In patients treated in hospital throughout an attack of faucial diph

theria peripheral neuritis is usually confined to the lower extremities and rarely amounts to more than paresis. The ankle- and knee-jerks are lost and may not reappear for many months. If the patient has been up and about his ataxic gait may draw attention to the condition. In such cases wasting, tenderness and paræsthesia are rare. Less commonly similar pareses affect the arms. Occasionally in neglected cases in adults complete paralysis of the muscles of one or other shoulder girdle occurs.

Very different in degree are the examples of polyneuritis associated with extra-faucial diphtheria observed by Walshe and others. A typical case recorded by Walshe is that of a soldier, who in May reported the sudden appearance of four septic sores on his hands and one on the left leg. The sores spread quickly and reached a diameter of  $1\frac{1}{2}$  inches. In late August the sores were still unhealed, and the man complained of numbness in the hands and feet and of constant tingling in the fingers and toes. By September 6th walking was difficult, there was weakness and aching of the legs, unsteadiness of gait and slight swelling round the ankles. Tenderness of the calves and a tendency to foot drop were noted and the tendon reflexes were absent in all four limbs together with sensory loss. In other cases paralysis of accommodation also occurred. Tenderness of the masseters, temporal muscles and weakness and tenderness to pressure of the sterno-mastoids may be demonstrated earlier than the generalised paralyses. Sometimes the syndrome of multiple neuritis first appears after unusual exertion, such as a route march, and may be associated with palpitation, shortness of breath, giddiness and faintness (Walshe).

The occurrence of any of the post-diphtheritic palsies is evidence of the fixation of toxin, and careful and repeated examinations of the heart are called for in order to detect damage as early as possible. Cardiac involvement may precede, accompany or follow the palsies, which in the latter case should be regarded as danger signals.

### Diagnosis.

The acute infective conditions which must be differentiated from faucial diphtheria are hæmolytic streptococcal tonsillitis and Vincent's angina. Hæmolytic streptococcal infections of the tonsils usually commence abruptly, possibly with shivering, nausea, and general aches and pains. The face is flushed, the temperature raised to  $102^{\circ}$  or  $103^{\circ}$  and the pulse rapid. The patient complains of sore throat, whereas in diphtheria this symptom is usually slight or



in 1 c.c.m. The advantages are obvious, greatly lessened incidence of serum phenomena, and the greater ease with which large doses can be injected without discomfort to the patient. It is claimed too that the pseudoglobulin-antitoxin molecule is smaller than in salt-precipitated serum and therefore that toxin absorption is more rapid. Although the real risks to the potential sensitive of serum shock (anaphylactoid reaction) are very slight it is wise, if possible, even before using the new serum to test for sensitivity and to desensitize such subjects, particularly asthmatics if necessary (*vide* page 298). The therapeutic serum (not normal horse serum) should be employed for the test and adrenaline should always be at hand. Ordinary serum sickness follows the use of these refined sera in about 5 per cent. of cases and is usually confined to an urticarial rash, readily controlled by adrenaline followed, if necessary, by ephedrine.

For cases of slight or moderate severity the intra muscular route (outer side of thigh) is adequate, but for severely toxic or late cases the whole or a portion of the dose must be injected *intravenously* in order to stem further damage by toxin forthwith. The subcutaneous injection of 4,000 to 16 000 units near the lesion is used by some for the treatment of desert sores. It is essential for patients in whom slow concentration in the blood-stream is necessary those refractory to desensitisation and asthmatics who must never receive serum intravenously.

It is impossible to lay down a really satisfactory scale of dosage it must be based upon a consideration of the extent and apparent age of the membrane, the local evidence of virulence (faucial hyperaemia, oedema and foetor adenitis and periaadenitis) and the existing degree of toxæmia. These factors also influence the route of injection. Any dose injected intravenously is equivalent therapeutically to double its size given intramuscularly and in adults intravenous injection is easy and free from risk if refined serum is used. For faucial diphtheria the dose varies from 8 000 to 16 000 or 20 000 units intramuscularly for cases of slight to moderate severity doses in excess of 20 000 units should be given in part at least intravenously. If there is no adequate therapeutic response in twelve hours additional antitoxin should be injected, preferably intravenously to make up for lost time. I have come to the conclusion that a dose in excess of 50 000 units of protein-digested antitoxic serum is very rarely called for provided that all-or part

sulphonamides for a variety of conditions agranulocytic angina (tonsillar sloughs and granulopenia) may have to be excluded. Diphtheria may be superimposed upon any of the foregoing conditions, and it is also liable to occur at some stage of an attack of enteric fever. A number of cases of combined paratyphoid A and faucial diphtheria came under my care during the last war.

*Bacteriological confirmation*—The use of the Folger-Solé swab (Solé, 1934, Parish, 1935) facilitates the bacteriological confirmation of diphtheria under field conditions. An ordinary throat-swab is dipped into horse serum (or antitoxin) and gently steamed over a flame until the serum is inspissated. The prepared swab after use is incubated for four hours (in the breast pocket in emergency) and then examined in stained smears in the usual way, sub-cultures being made if feasible or desirable. The diphtheria bacillus is detected with greater ease and certainty than in smears made from ordinary swabs. Hæmolytic streptococci are readily identified if a swabbing is plated in blood-agar and incubated overnight. Naked-eye examination in the morning shows the unmistakable colourless hæmolysed zones around each colony. Vincent's organisms are readily seen in smears stained with dilute carbol-fuchsin or with Leishman's stain.

*Post-diphtheritic stage*—The man may first report sick for breathlessness and giddiness on exertion or weakness and discomfort in the limbs or both combined. The cardiac condition may be due to "effort syndrome" or to beri-beri. The former is not associated with peripheral neuritis. Beri-beri presents a combination of tachycardia and peripheral neuritis, associated or not with peripheral or generalised œdema, but the central palsies of accommodation and palatal paralysis do not occur.

### Treatment.

The essentials of treatment consist in antitoxin therapy at the earliest opportunity, the enforcement of complete and prolonged rest, and accessory measures dictated by the occasion, e.g. glucose therapy and, very rarely in the adult, operative procedures for the relief of laryngeal obstruction.

*Antitoxin*—In civil practice and doubtless whenever possible in the Services protein-digested (globulin-modified) serum is now used. This product is virtually protein-free and contains a high unitage of antitoxin in a very small volume of fluid, e.g. 6,000 units or more

In most cases bacteriological clearance is obtainable by or before the patient is clinically fit. Hartley and Martin (1919) showed that the rate of clearance among a group of Army patients during the last war could be expressed by a logarithmic curve.

A few patients remain persistent carriers, they usually have some unhealthy or abnormal condition of the naso-pharyngeal mucosa. Provided the patient has abundance of fresh air the carrier state may clear spontaneously. Antiseptic gargles are of little value for the tonsillar carrier who may ultimately require tonsillectomy—an almost certain cure. The nasal carrier is more troublesome obvious abnormalities such as polypi, spurs or enlarged turbinates must be dealt with. Chronic nasal carriers frequently have a double infection of diphtheria bacilli and *hemolytic streptococci* which responds well to insufflations of sulphanilamide powder (Boussard and Fry, 1942).

The bacteriological criteria of clearance adopted for the persistent carrier should be more rigorous than for the ordinary case. Three consecutive platings upon tellurite media are desirable and it is important always to establish the virulence of the strain, because virulence may disappear and there is no justification for the further detention of the carrier of avirulent organisms.

Prophylaxis consists in the prompt segregation of the actual or suspected case the search for carriers in the immediate environment of the patient and their segregation, the daily observation (but no isolation, for a week of contacts, and attention to the condition of the sleeping quarters with special reference to space between heads ventilation and the reduction of dust. A combination of Schick testing and swabbing is of great assistance in the detection of carriers chronic carriers are Schick immune. If thought desirable, the positive reactors may be passively immunised 1 000–2 000 units of antitoxin will protect them for three weeks. It is better still to combine passive with active immunisation and to inject also a first dose of alum precipitated toxoid (A.P.T.) the second dose following a month later. The reactions with current preparations and A.P.T. are rarely severe in adults.

The active immunisation of recruits against diphtheria as a general prophylactic measure is a question of policy upon which any discussion here would be out of place. For some years recruits in the French army have been compulsorily inoculated simultaneously against diphtheria tetanus and the enteric fevers (Ramon,

of the dose is given intravenously. Extra-faecial types of diphtheria respond to doses of from 4,000 to 16,000 units

*Glucose*—In the severely toxic case the intravenous injection, repeated if necessary, of 20 grams of glucose in a 40 or 50 per cent solution (the 10 per cent "resuscitation" solution involves the introduction of too much fluid) may avert circulatory failure, and it is in my experience the only likely means of restoring the failing heart, either early or late. The ordinary cardiac drugs are of little value in diphtheria, nikethamide (coramine) is the most useful. Less severe cases benefit from four to six ounces and upwards of glucose by the mouth on each of the first ten days of the illness.

*Rest*—Rest in bed, which implies posture and movement, is of great importance. Subject to medical discretion in the individual case the following well-tried routine should be followed: flat for a week and thereafter a pillow a week up to three, then for a week gradually increasing periods on a couch. Finally the patient is dressed and allowed progressive periods of gentle exercise until cardiac and general muscular tone are restored. During the first two weeks movements should be passive only. The occurrence at any stage of a post-diphtheritic complication is an indication that the scheme should start afresh. It is a great mistake to hurry the convalescence of the diphtheria patient. Occasionally the restoration of function of the limbs after peripheral neuritis may necessitate gentle massage and rarely faradisation.

*Criteria for release from hospital*—As always in infectious diseases there are two criteria for release from isolation, viz. clinical fitness and freedom from infection. Except that a few cases of permanent heart block are on record and that the rare post-diphtheritic hemiplegia leaves residual effects, the complications of diphtheria, if they do not kill, at length clear up completely, although many weeks may pass before this occurs. It should be noted that the knee-jerks once lost may not reappear for months, but provided the patient's condition is otherwise normal, this alone is not a cause for detention. The Services have their own standards of bacteriological control; in civil practice it is customary to obtain two or three consecutive negative cultures from the throat and nose before release from isolation. Swabbings should be made at intervals of not less than five days, and care must be taken that no antiseptic gargle has been used within some hours of swabbing.

In most cases bacteriological clearance is obtainable by or before the patient is clinically fit. Hartley and Martin (1919) showed that the rate of clearance among a group of Army patients during the last war could be expressed by a logarithmic curve.

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1939) A new formula for the triple prophylactic has recently been introduced and is said to cause fewer severe reactions than the original mixture (Ramon, 1942)

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## CHAPTER XXVI

### TETANUS

The danger of tetanus as a complication of wounds has been recognised since the days of Hippocrates, and little can be added to the classical descriptions of the acute disease. The main facts concerning the pathology of tetanus were discovered between 1885 when Nicolaier produced the disease in mice by inoculating them with garden soil and isolated a characteristic bacillus from the local lesion, and 1890 when Kitasato having prepared in a pure form the toxin which Faber had discovered the previous year laid the foundation of serum prophylaxis and treatment by showing that animals could be rendered immune by the repeated injection of non lethal doses of the toxin, and that their serum had the property of neutralising it *in vitro*. But tetanus deserves a special chapter in a book on medical diseases of war as the war of 1914-18 was the means of at last demonstrating beyond doubt the value of antitetanic serum in prophylaxis. It showed how passive immunisation diminishes the severity of the disease and modifies it in such a way that the majority of cases show premonitory symptoms, which were formerly scarcely recognised but which now enable treatment to be instituted so early that the chances of success are greatly increased, and that in other cases the symptoms remain localised, giving rise to a remarkable form of tetanus, which had never before been described. The present war has already proved the great value of active immunisation.

#### *Ætiology*

Tetanus bacilli and their spores do not inhabit uncontaminated soil, but are frequently present in the intestinal contents of horses and cattle. As the spores are exceedingly resistant, soil once contaminated with horse- or cattle-dung continues to be a source of danger for an indefinite period. As cattle and horses are much more numerous in some districts than in others, and the frequency with which tetanus bacilli are present in their faeces varies greatly in different localities, the danger of contamination of wounds with soil is very inconstant. Thus in spite of the absence of prophylactic inoculation the disease occurred in only 508 or 0.2 per cent., out of 246 712 wounded men in the American Civil War and it was so

rare in the South African War that Makins only heard of a single case. On the other hand, there is little doubt that it would have been extremely common in France and Flanders had not universal prophylactic inoculations been introduced at an early date. The greater frequency of tetanus after wounds received at the battles of the Marne and Aisne than in the neighbourhood of Ypres was doubtless due to a corresponding difference in the number of tetanus spores in the soil, although Robertson (1918) found that the soil in all parts of the Western Front was so heavily contaminated that inoculation of a single grain into a laboratory animal invariably caused tetanus. The rich soil in the valley of the Aisne has such an evil reputation for tetanus that the farm-horses had for several years before the war been periodically inoculated with prophylactic doses of antitoxin. Tetanus was very rare on all other fronts. There were only six cases in Gallipoli, all in patients with trench feet, four in Salonica and seven in Mesopotamia.

The risk of developing tetanus in the Middle East is very much less than in France and Flanders, though the main tracks are extensively contaminated with the desiccated droppings of donkeys, ponies and camels, as the dry sandy soil of the desert is unsuited for the propagation of anaerobes. Tetanus spores were found in only 8.8 per cent of samples of soil obtained from ninety-one different spots between Daba and Benghazi, the majority being from the more fertile country west of Tobruk. In spite of the rarity of tetanus spores in the soil they were found in 8.4 per cent of cultures obtained from 214 severe wounds, in most of which gas gangrene was present (Boyd and MacLennan, 1942).

The tetanus bacillus may enter a wound the moment it is inflicted, or the infection may occur owing to the wound becoming soiled by earth when the soldier falls or crawls to a place of safety. A bullet, being sterile, is not likely to cause tetanus except after a ricochet. Tetanus spores are much more frequently introduced by shrapnel, as the shell bursts on the ground so that its fragments are likely to carry earth with them. Moreover, fragments of shell are likely to carry dirty pieces of clothing with them, whereas a pointed bullet often makes a clean hole through the uniform. Tetanus is most frequent when heavy fighting occurs in wet weather, when the mud leads to severe infection of wounds.

In all cases the wounds had been septic at some period, but they had often almost or completely healed by the time tetanus

developed. They might be severe or trivial but the risk is greater the larger and more septic the wound. Culturally *Cl. tetani* is a strict anaerobe. Small quantities of oxygen prevent its growth. Injected, washed or toxin free spores remain dormant in healthy tissue, whereas if they are placed in damaged tissue or accompanied by traces of toxin, ionisable salts of calcium, particles of soil, or sepsis-producing bacteria, they soon germinate. This germination first takes place when the local necrosis produced by injury or by the above substances results in sufficient lowering of the oxygen tension. As soon as the tetanus spores germinate, further multiplication and the local production and absorption of tetanus toxin begins. Hence the liability of tetanus is greatest in lacerated and contused wounds, in cases of compound fracture and in wounds in which a foreign body is present.

In the American Civil War 26 per cent. of the cases of tetanus followed immediately after a secondary operation. In the last war also the appearance of tetanus was frequently delayed for weeks or even months after a wound had been inflicted, when a secondary operation, often of a quite trivial nature, at the site of the original wound, even if it had completely healed was followed a few days later by the appearance of tetanus.

A considerable number of cases of tetanus developed in France as a complication of trench foot, and I saw the six cases in which this occurred at Lemnos after the great storm at Gallipoli at the end of November 1915\*. The oedema fluid infiltrating the connective tissue of the foot afforded a perfect culture medium for the growth of the tetanus bacillus, and the chilling of the tissues inhibited the leucocytes from taking up the spores. Tetanus also followed septic burns in one case the man had rolled in the mud in order to extinguish the flames.

#### Incubation Period

The incubation period of tetanus in the last war varied between 2 and 365 days. The average in cases occurring in France was twelve days, the largest number developing on the eighth day three days earlier than in the cases occurring in England (Leishman and Smallman, 1917). The incubation period became longer when the vast majority of wounded soldiers received prophylactic injections. In the first year of war 47 per cent. of cases but between December

\* *Vide* an Editorial in the *Jour. R. Army med. Ops.*, 1940 74 154 for an admirable account of Trench Foot.

1916 and March 1917 only 10 per cent, had an incubation period of less than ten days (Bruce). An incubation period of more than twenty-two days occurred in 69 per cent of cases in a later series, but in only 6.4 per cent in the first year and in 5.7 per cent in the Franco-Prussian War.

When symptoms do not appear until three weeks or longer after the wound, the true incubation period is probably much shorter than it appears to be. The *Cl. tetani* is generally, if not always, introduced into wounds in the form of spores, which do not themselves produce toxin. The spores may never germinate, or they may produce the bacillus only after an interval of days, weeks or months, during which the wound may have completely healed. In many cases of this sort the true incubation period dates from a secondary operation, which has had the effect of stimulating the spores to develop into bacilli, which at once begin to produce toxin.

### The Cause of Symptoms.

The toxin produced by tetanus bacilli in a wound passes into the blood and lymph, in which it can actually be found both in animals and man, the amount reaching a maximum at the time the first symptoms appear. Part of the toxin is carried to the central nervous system by the motor nerves, the nerve endings in the neighbourhood of the wound absorbing it from the surrounding lymph. The first part of the central nervous system to be involved is consequently the segment connected with the motor nerves in the neighbourhood of the wound. From this situation the toxin spreads along the spinal cord to the brain, except in the case of wounds of the face, when the toxin passes direct to the pons and medulla by the facial nerve. At the same time some of the toxin circulating in the general blood-stream reaches the central nervous system without passing up the motor nerves.

The toxin, having entered the spinal cord or brain, combines with the nerve-cells. It renders them abnormally sensitive to all stimuli, so that the afferent impulses which are constantly reaching them produce a tonic contraction of the muscles they supply, and every additional afferent impulse causes an additional spasm. In unprotected men and in large animals, such as horses, the toxin which reaches the medulla and pons from the blood produces trismus, which is followed by other symptoms as other centres become affected.

In the cat, which is relatively insusceptible to the tetanus toxin,

a non-lethal dose leads to very persistent local spasms of the limb into which the toxin was injected, but no generalised contractions occur as the only toxin to take effect is that which reaches the spinal cord by the motor nerves. If in the more susceptible guinea-pig a sufficient quantity of antitoxin is injected with the toxin or shortly afterwards, the result is the same. Local tetanus hardly ever occurs in uninoculated man, as the susceptible centres in the pons and medulla are attacked before the spinal centres. But antitoxin has the same effect in man as in the guinea pig. Any toxin circulating in the blood is neutralised by the antitoxin introduced by prophylactic inoculation. The centres in the pons and medulla are thus protected, as they are not affected by the toxin ascending the motor nerves, except the facial nerve in the case of wounds of the face. But the spinal centres may be attacked, as they are reached directly by the toxin travelling up the nerves from the wound. The spasms therefore generally begin in the neighbouring muscles. If insufficient serum is given, the protection afforded by the antitoxin gradually disappears and the pontine and medullary centres may finally be attacked. When, however sufficient serum has been injected, no toxin reaches the pons and medulla, and the symptoms remain localised. The spasms may spread to the muscles of the rest of the limb or even to those of the opposite side and the neighbouring part of the trunk, as they receive their nerve supply from adjacent parts of the spinal cord, into which the toxin can readily spread.

The muscles at first relax under the influence of a general or intraspinal anæsthetic, which depresses the abnormal excitability of the nerve-centres. But after about thirty hours the muscles themselves are altered in such a way that the spasm persists under these conditions, and in animals it is not completely abolished by dividing the motor nerves or even excising pieces of the affected muscles.

Generalised spasms appear in men who have received protective inoculation only under two conditions—such an overwhelming quantity of toxin may be absorbed that the antitoxin cannot neutralise it, and generalised tetanus occurs after a short latent period—or toxin may be formed by bacilli, which develop from spores after all the antitoxin has disappeared from the circulation, but the latent period is then prolonged.

### Premonitory Symptoms.

In his description of the tetanus which occurred in the Armée d'Orient in Egypt and Syria in 1803, Larrey wrote " Cette maladie commence par un malaise générale et une sorte d'inquiétude qui s'empare du blessé . . Ce phénomène est accompagné de douleurs aiguës, qui augmente par le contact de l'air et des plus légers corps extérieurs , la totalité du membre devient douloureuse. L'irritation musculaire s'étend rapidement des muscles voisins de la plaie, aux plus éloignés qui se contractent , ou bien elle se transporte tout-à-coup aux muscles de la gorge et des mâchoires où elle se concentre "

Although cases with a similar history were collected by Poland from the Guy's records between 1820 and 1857, very little attention was paid by subsequent writers to these premonitory signs, the great importance of which became recognised only after the beginning of the last war.

The occurrence of pain in the neighbourhood of a wound is so common that it cannot by itself be regarded as of any importance. But if a patient, who has been quite comfortable for some days, suddenly develops a sharp recurring pain in the neighbourhood of the wound without obvious reason and without any change in the appearance of the wound, the possibility of tetanus should be considered. In several instances this premonitory pain has been regarded as rheumatic. The surrounding muscles, which were previously soft and relaxed, may appear to be abnormally rigid, and exaggerated irritability of the muscles is shown by their spasmodic contraction on the slightest stimulation.

Scattered through the literature of tetanus are references to cases which began with spasms in the wounded limb. Key in 1836 and Fagge in 1886 described cases they had seen at Guy's, in which the local spasms were throughout the most prominent feature of the illness. Now that all wounded men receive prophylactic injections, the first symptom is local in about half of the cases in which the disease finally become generalised. Some of the muscles around the wound twitch or show irregular clonic or tonic contractions, or tonic contractions may occur in a group of muscles nearby, especially in the flexors. These symptoms may persist for a few hours up to twenty-five days before the jaws become involved. Andrewes (1917) recorded the case of an officer, who received an injection of antitoxin six hours after being wounded

in the thigh. Nine weeks later, when the wound was healed though a small fragment of shrapnel was still present the leg felt rather stiff when he walked. In spite of this he returned to duty in France and remained at his work for a fortnight, when the stiffness was so great that he could no longer walk. Trismus, stiff neck, and opisthotonos then developed, but recovery followed the daily intrathecal injection of large doses of antitoxin for eighteen days.

Other symptoms occasionally precede the development of tetanus, probably as a result of the general toxic action of the tetanus poison. Outbursts of temper with unusual restlessness and insomnia occur, and a patient, who was previously feeling very well, may complain of severe headache and giddiness. Excessive yawning and slight difficulty in micturition have also been observed.

### Symptoms

Apart from the muscles in the neighbourhood of the wound the earliest to be involved are the masseters and posterior muscles of the neck. The patient generally notices the stiffness of his jaws for the first time when he wakes in the morning. The spasm increases until it is impossible to force the jaw open. At a later stage the face becomes affected—the lips are stretched over the clenched teeth so as to produce a fixed and mirthless smile—the *risus sardonicus*—the naso-labial folds are exaggerated, the forehead is wrinkled and the eyelids half closed, but the muscles of the eyes and tongue are not involved (Fig 42).

I saw an officer in 1915 who had had his jaw shattered by a piece of shrapnel so severely that the dental surgeon in charge found it impossible to fix the fragments. On the sixth day he developed tetanus, and the contracted masseters acted as a splint so efficiently that when he recovered from the tetanus the fragments of the jaw had united firmly in excellent position, and there was no obvious deformity except a depressed scar where the fragment of shrapnel had entered.

Before any definite retraction is present it is often found that the head cannot be bent sufficiently forward for the chin to touch the chest. The recti abdominis muscles are next involved, and then the other muscles of the abdomen and back. In the fully developed disease the head is retracted and the back is arched. Arctans described a form of tetanus under the name of *emprosthotonos*, in which the body is bent forward instead of backwards as in the familiar *opisthotonos*, but this, like *pleurosthotonos*, in

which the body is bent to one side owing to the asymmetrical distribution of the spasm, is extremely rare. The muscles of the legs, especially the flexors of the hips and knees, together with those of the upper arm, take part in the general spasms, but the forearms and hands are spared.

In addition to continuous tonic contractions of the affected

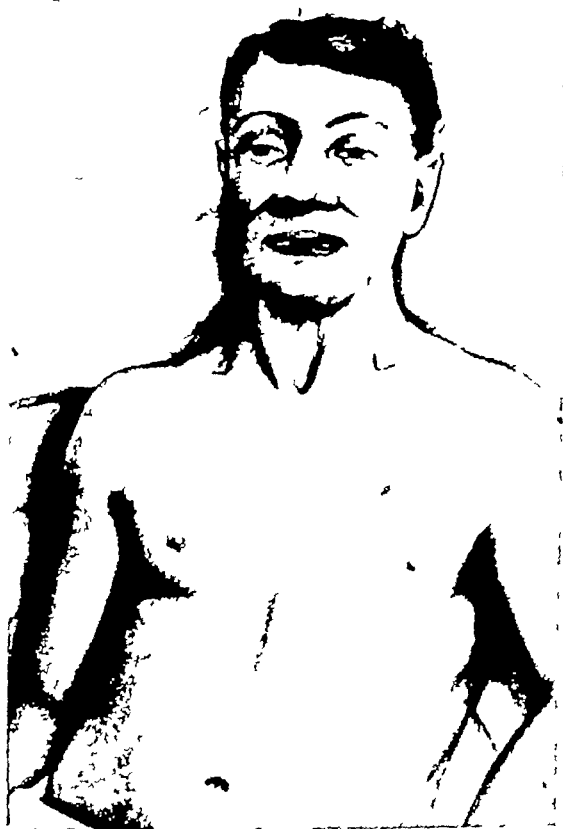


FIG 42 —Generalised tetanus following the infection of a blister on the foot. The characteristic facies and general rigidity are shown. The recti are rigid, but the abdominal wall is flat. (*Reproduced by permission from a drawing in the war collection of the Royal College of Surgeons*)

muscles, tonic and clonic spasms occur. They may appear to be spontaneous, but more frequently they are obviously reflex in origin and follow a touch, exposure to a draught, a bright light or a sudden noise.

Sherrington (1917) demonstrated in animals that the tetanus toxin disturbs reciprocal innervation. Thus stimulation of the cerebral

cortex in the area, which should cause the mouth to open by contraction of the depressors and relaxation of the elevators of the jaw produces trismus, as the normal relaxation of the elevators, which are stronger than the depressors, is converted into contraction. Similarly in man an effort to open the mouth, instead of causing relaxation of the masseters at the same time as the depressors contract, leads to their contraction, the trismus being thereby increased. In the same way voluntary efforts to move other affected parts increase the spasm. Thus apart from the difficulty in taking food caused by the trismus, an attempt to swallow often causes spasm of the pharyngeal muscles.

The constant tonic contractions make the muscles ache, and the spasms cause a varying amount of pain, which often prevents sleep. More or less complete relaxation may be produced by drugs, which in this way make sleep possible, but the spasms quickly return a few minutes after waking.

The increased excitability of the nervous system is shown by the exaggerated tendon and bone reflexes. Thus the wrist- and knee-jerks are increased, ankle-clonus may be present, and the jaw jerk is obtained with unusual ease, even in local tetanus involving a leg.

When the spasms are severe, the pulse becomes rapid and sweating is profuse, but the temperature may remain normal, especially if the wound has already healed. A pulse of 100 to 130 with a normal temperature is characteristic.

Even in the severest cases the mind remains clear until the end, but in rare instances delirious and hallucinations occur.

Death may result from asphyxia caused by prolonged spasm of the respiratory muscles, especially the glottis. Sudden or gradual heart failure may occur, generally after the pulse has been very rapid for a time. If death takes place in the later stages, when trismus is diminishing and spasms have become rare or have even ceased, it is generally due to the exhaustion caused by toxæmia and malnutrition. Occasionally extreme hyperpyrexia develops shortly before death. Septic pneumonia, which may follow aspiration of food, and oedema of the lungs are very fatal complications.

Recurrence may follow apparent recovery. In one case, which began on the sixteenth day after the wound was received, the patient had apparently recovered by the twenty-fifth day but on the forty-second day the symptoms returned and he died on the seventy-fourth day (Westwater 1917).

The cerebro-spinal fluid is normal, but in severe cases its pressure is raised. It may contain the tetanus toxin, but Sir Charles Sherrington found that injection of the fluid in three of my cases of localised tetanus produced no symptoms in cats

### Diagnosis.

Early diagnosis is essential for successful treatment. The possibility of tetanus should be constantly borne in mind, and the occurrence of unexplained spasms of pain, "rheumatism," or twitching of muscles in the neighbourhood of a wound should immediately arouse suspicion. Any hardening of the muscles, either spontaneously or on pressure, and exaggeration of the tendon and bone reflexes, especially of the wounded limb, would render tetanus still more probable. The slightest stiffness or aching of the jaws or the back of the neck should also at once suggest the possibility of tetanus.

The diagnosis must be founded upon the clinical features of the case. It is a serious mistake to wait for the discovery of the characteristic drumstick bacilli in the discharge from the wound before injecting serum, as in many cases repeated examination fails to reveal the organism in undoubted cases of tetanus. Conclusive evidence is afforded by the production of tetanus in animals by the subcutaneous injection of the discharge from the wound, or, if this fails, of the filtrate of anaerobic fluid cultures. But such tests are valueless as a guide to treatment, as the incubation period of experimental tetanus is between three and four days.

Even in the earliest stages there should never be any difficulty in differentiating trismus due to tetanus from that produced reflexly by a dental abscess or an impacted wisdom tooth, or directly by inflammation of the temporo-maxillary joint or parotitis. A careful examination of the masseters and the neighbouring structures, the history of the onset, and the very early presence of slight spasm of the posterior cervical muscles in tetanus should prevent the possibility of a mistake.

I once saw a case of hysterical trismus, which was at first diagnosed as tetanus. A man had trodden with his bare foot on the point of a rusty nail, and two days later he developed well-marked trismus. I was asked to see him in order to advise how much serum should be injected. The very short latent period, however, aroused my suspicions, and I inquired whether he had ever known or heard of anybody having lockjaw. He told me that he had read in the

paper on the previous Sunday how a man had died of lockjaw after an accident precisely similar to his own. I explained to him that his lockjaw was the kind that always got well in a few hours he was already much better an hour later and by the next morning the spasms had completely disappeared.

### Prognosis

In the American Civil War the mortality of tetanus was 89·3 per cent., and in the Franco-Prussian War it was 90 per cent. among the German troops. In civil practice the average mortality before the introduction of serum for prophylaxis and treatment was about 86 per cent. In the last war the mortality became steadily smaller as prophylactic injections of serum became more universally adopted. It was difficult to say to what extent treatment with serum helped to reduce the mortality but there could be no doubt that improved methods of treating septic wounds and earlier diagnosis were important factors. In the 231 cases occurring in England during the first year of the last war the mortality was 57·7 per cent. and that of the next 195 which occurred up to the end of July 1916 was 49·2 per cent. between August and October 1916 there were 200 cases with a mortality of 38·5 per cent. and between December 1916 and March 1917 100 cases with a mortality of only 19 per cent. which would, however be raised to 28·3 per cent. if the 19 cases of localised tetanus, in which there was no mortality were excluded (Bruce)

The earlier the prophylactic dose of serum is given the greater is the chance of recovery and with the modern method of active immunisation there should be a very low mortality among the few cases occurring in protected individuals.

The mortality is greater with a short than with a long incubation period. The average incubation period in 115 fatal cases occurring in France was 10·7 days that in the 42 cases of the same series which recovered was 14 days (Lousman and Smallman). In Bruce's last series of 100 cases occurring in England the mortality was 40 per cent. when the incubation period was ten days or less, 25 per cent. when it was between eleven and twenty four days, and only 13·6 per cent. when it exceeded twenty five days. Tetanus, however is not without danger whatever the incubation period one patient died, although his symptoms did not appear until seventy five days after he was wounded (Miller 1917). But every day the patient survives after the sixth the more hopeful is the outlook.

The prognosis also varies with the interval between the occurrence of the first symptom and the appearance of generalised reflex spasms—"the period of onset" (Cole, 1935). As a rule the longer the incubation period the longer the period of onset, but when the latter is shorter than would have been expected from the length of the former, the period of onset gives the better indication of the severity of the case. In well-treated cases recovery generally occurs with a period of onset exceeding two days, rarely if it is less than two days (Cole). Death is very unusual in chronic cases, in which reflex spasms do not occur at all or only a week or ten days after the first symptom.

The prognosis is worst with extensive, severe, multiple or very septic wounds. When tetanus is complicated by gas gangrene, as it frequently was in France, the mortality is very high, this being the chief cause of the much greater death-rate among British troops in France than in England, but the shorter incubation period was an important additional factor. The mortality in England could be much more fairly regarded as the true mortality of tetanus with modern methods of prophylaxis and treatment than that in France, in which death was frequently due in part, if not entirely, to the severe nature of the wound itself or to septic complications.

The prognosis is worst when successive groups of muscles are affected in rapid succession, as happens especially when the incubation period is short. Very severe and prolonged reflex spasms are likely to lead to death from asphyxia unless they are controlled. Stridor, indicating spasm of the glottis, a temperature over 104° F before the fifth day, and hyperpyrexia at any time are bad signs.

### Prophylaxis.

#### *Passive Immunisation*

I have already described how the mortality of tetanus fell as the incubation period became longer, and the relative frequency of localised tetanus greatly increased as a result of prophylactic injections of antitetanic serum. The symptoms also developed much more slowly. The following statistics show how the incidence of the disease at the same time was greatly reduced. The proportion of cases of tetanus to the total number of wounded British troops arriving in England reached the high figures of 15.9 per 1,000 in September 1914 and 31.8 in October. The incidence at once fell when prophylactic injections of serum began to be used on a large scale in the middle of October: 600 doses had been sent to France

in August 1914 and 12,000 in September, but 44 000 were sent in October 112,000 in November and 120 000 in December. Only 1·7 cases per 1 000 wounded occurred in November and 0·9 per 1 000 in December 1914. The incidence then remained constant but during the last year of the war it fell still further owing to the more universal use of serum for even trivial wounds and to the greater frequency with which repeated injections were given in septic wounds and additional injections were given before secondary operations.

Similar reports were published by French and German observers. Bary gave a striking example of the value of prophylactic inoculations. Owing to an accident only 100 out of a batch of 200 wounded men were given serum. Among the 100 who had been inoculated the only one who developed tetanus did so on the day of inoculation before the serum had a chance of exerting any prophylactic action, whereas eighteen cases occurred among the uninoculated 100 although their wounds were no more severe or septic.

The first dose of 3 000 units\* should be given at the earliest possible moment after the wound is received. As tetanus toxin does not lead to any appreciable degree of active immunity any further toxin produced by tetanus bacilli still in the wound is free to act after the initial prophylactic dose of antitoxin has disappeared from the circulation, unless the formation of a granulation-tissue barrier prevents its absorption. A second subcutaneous injection of 1,000 units should therefore be given in all cases of septic wounds seven days after the first injection in order to anticipate the disappearance of the antitoxin from the body about the tenth day and the consequent loss of immunity. The dose at present recommended by the Army authorities and the Ministry of Health is three injections of 3 000 I.U. given at weekly intervals. This should be done however trivial the wound may appear to be as in at least five out of twenty-five cases of tetanus seen in England by Dean (1917) the wounds were so slight that the most conscientious medical officer would hardly have thought of giving a second prophylactic injection. In chronic septic wounds especially those caused by shells or bombs three, four or even more numerous injections should be given at seven-day intervals. The danger of anaphylaxis with an initial

\* The international unit (I.U.) is now used in this country instead of the American unit (A.U.), which is double the strength. The latter which is still employed in the United States, was in use in the British Army during the last war.

prophylactic dose of 3,000 and later doses of 1,000 units given subcutaneously was found in the last war to be quite negligible, whatever the interval might be between the injections

If more than a week has elapsed since the last dose of antitoxin was given, an additional 3,000 units should be injected intravenously whenever an operation is to be performed in the neighbourhood of a wound, even if it has completely healed.

Prophylactic injections of antitetanic serum should also be given in all cases of severe burns and in trench foot, even when the skin is unbroken. The danger of tetanus as a complication is almost completely abolished when this is done

### *Active Immunisation*

Active immunity against tetanus became possible after the discovery by Ramon in 1924 that toxin, treated with a low concentration of formalin and kept at a temperature of 37° C for a month, loses its toxic action but retains its antigenic proportions. The tetanus "toxoid" produced in this way gives rise to no ill-effect on injecting into animals and produces satisfactory immunisation in laboratory animals and horses. In 1927 Ramon and Zorller found that the toxoid was equally effective in man.

The first injection of tetanus toxoid produces little or no antitoxin, but it leads to sensitisation, probably of the reticulo-endothelial system, with the result that a second injection after a suitable interval is followed by the immediate vigorous production of antitoxin. Boyd (1938) has shown that after an interval of six weeks the second injection produces an antitoxin titre as great as that obtained when an additional intermediate dose is used, as originally recommended by Ramon. The routine method now in use in the British Army is the subcutaneous injection of two doses of 1 c cm at an interval of six weeks, with additional doses every twelve months. In the American Army active immunisation is compulsory and toxoid is given every four months whilst a soldier is on active service.

Prophylactic shock after injection of tetanus toxoid is very rare, but not unknown. It is caused by the peptone of the medium in which the toxoid is prepared. Whittingham (1940) recorded two cases of severe shock and fourteen of milder symptoms developing within an hour of the second injection of 1 c cm given six weeks after the first. These were the only cases among 61,042

men in the R.A.F. who were inoculated this gives an incidence of 0.003 per cent. of severe and 0.023 per cent. of mild symptoms. The double inoculation leads to the production of 0.1 to 1.0 units of antitoxin per c.cm. in the recipient's blood. The sensitisation produced by the first dose has the further effect of leading to the production of antitoxin in response to any toxin introduced as a result of infection with the tetanus bacillus. The curve of antitoxin production probably exceeds that of toxin absorbed from the wound so that effective neutralisation results. Immunisation lasts for a considerable period. At the end of a year the concentration of antitoxin in the serum shows some diminution, but there is little more diminution in two years and even after five years it remains well above the level necessary to confer immunity against infection. Moreover after five years the serum still responds to a further injection of toxoid by the immediate production of antitoxin, so that permanent immunity could probably be maintained by inoculation of toxoid repeated every five years.

In the present war 90 per cent. of the British Army are protected by active immunisation with tetanus toxoid, and every casualty receives 3 000 units of antitoxin as soon as possible after being wounded. American and Canadian soldiers, however are given an additional dose of toxoid if wounded, serum being reserved for the treatment of actual tetanus. During the evacuation from Dunkirk it was often impossible to administer the antitoxin, though many men were inoculated on reaching England. There were no cases of tetanus among the inoculated, and only eight among the uninoculated, making a total of 0.45 per 1 000 wounded. In one of these cases the explosion of a tin of petrol caused facial abrasions. Twelve days later tetanus developed. The patient recovered after being given serum intravenously. He was one of the few soldiers who had not been immunised with tetanus toxoid, and he was not given antitetanic serum when admitted to hospital on the day of the accident (Medicus, M.P. 1940). No case of tetanus occurred among the 600 Canadian soldiers wounded in the Dieppe raid. All had been immunised with tetanus toxoid, but toxoid or serum was given in only a very small proportion after wounding (MacFarlane, 1943).

The incidence of tetanus among wounded soldiers from Great Britain, India and New Zealand in the Middle East was 0.13 per 1 000 but among the South Africans, who had not been immunised,

it was 1 6 Of the eighteen cases occurring between January 1941 and July 1942 five were in men who had been actively immunised, four with two and one with three doses, but none had received the routine dose of 3,000 units of antitetanic serum after being wounded. Three of the five died. In two of these there was extensive necrotic tissue in the wound, which must have led to an overwhelming infection. This should have been prevented with adequate surgical treatment by the removal of all dead tissue. The remaining thirteen cases, six of which were fatal, occurred among the relatively very small number of men who had not received active immunisation.

In civil practice patients have not already been immunised, so the importance of passive immunisation remains as great as ever. In such cases French authorities recommend the immediate injection of 1 c cm. of tetanus toxoid and 3,000 units of antitetanic serum, followed by two further injections of toxoid at fifteen-day intervals.

### Treatment.

*Antitetanic serum*—When symptoms of tetanus appear, some toxin is present in the nerve-cells, some is still circulating in the blood, and a further quantity is probably still being formed by the tetanus bacilli in the wound, from which it continues to be absorbed. The object of treatment by antitoxin is to neutralise and destroy at once the toxin present in the circulation at the time of inoculation, and to provide for the presence of a sufficient quantity in the blood to neutralise further toxin directly it is absorbed from the wound. It is theoretically possible that some of the toxin actually combined in the nerve-cells may be dissociated if the concentration of antitoxin in the surrounding fluid is sufficiently great, but it is doubtful whether this object can ever be attained in practice.

Experimentally the intravenous and intrathecal administration of serum is much more effective than subcutaneous and intramuscular injection, and in practice the latter are painful and absorption is slow. Theoretically intrathecal injection might not be expected to be of much value, but experimentally it has been found to be very effective. As, however, the injection of a foreign protein sometimes leads to meningeal irritation with pain and stiffness in the back and excess of polymorphonuclear cells in the cerebrospinal fluid, and as it is much less convenient than intravenous injection in patients suffering from tetanus, its use should be

abandoned, in view of the fact that there is no clinical evidence that its use either instead of or in addition to intravenous injection gives any better results than intravenous injection alone. When a sufficiently large dose is given intravenously excellent results are obtained. Thus thirteen out of Dean's fourteen cases of generalised tetanus treated by a single large intravenous injection during the last war recovered. The single death occurred whilst the injection was being given under an anaesthetic not from anaphylaxis. Thus there was no death among cases in which the serum had an opportunity of acting. Six patients, who received one or more additional doses by intramuscular or subcutaneous injection improved no more rapidly than the seven, who each received a single intravenous injection alone. Further evidence in favour of a single large intravenous injection is afforded by the series of forty cases treated between 1930 and 1938 by Cole. None had received protective inoculation and the mortality was 47.5 per cent. If six patients over 60 and five who died within 24 hours of admission are excluded, the mortality was only 28 per cent.

A single large intravenous dose should be given directly the diagnosis is made in order that all the free toxin in the body may be neutralised, and in the hope that some which has already reached the nerve cells may be dissociated. No immediate improvement can be expected. The best that can be hoped for is that no more nerve-cells will be involved. The recovery of those already affected leads to slow improvement, which may manifest itself in forty-eight hours by a fall in the pulse-rate, although diminution in the spasms is rarely obvious before the fourth and sometimes not until the seventh day.

During the last war Dean recommended an intravenous dose of 60 000 units, but Cole has shown that a much larger quantity is desirable and recommends 200 000 units as a routine. Dean found that the blood still contained antitoxin twenty thirty and even thirty nine days later although no further serum had been injected. In four of Cole's cases there were still 50 000 units in the circulating blood ten days after the injection of 200 000 units and about 25 000 units fourteen days after. As a prophylactic dose of 3 000 units is generally sufficient to protect even with a severe and badly infected wound, it is clear that nothing can be gained by giving repeated injections.

The danger of anaphylaxis, which is almost negligible with

subcutaneous and intramuscular injections and very slight with intrathecal injections, is undoubtedly present when the intravenous method is used, but with proper precautions it can be minimised. Cases of anaphylaxis were extremely rare in the last war, in spite of the fact that the vast majority of men who developed tetanus were hypersensitive owing to having already received a prophylactic dose of serum a short time before. The precautions already described in connection with anti-dysenteric serum should be used in all cases (p 298).

*Drug treatment*—The best drug for the prevention of spasm, relief of pain and inducing sleep is avertin. It was first used in tetanus by Momburg and Rotthaus (1929), and it has proved of great value. The dose is the same as for basal anaesthesia—0.1 c cm per kilo-body-weight. The avertin is given at first every six or eight hours according to the severity of the spasms and the effect of the previous dose. As the spasms become less frequent it is given at longer intervals, but in severe cases it may be necessary to continue for several days. In mild cases, in which reflex spasms are short and infrequent, 4 to 6 dr of paraldehyde may be given by rectum (1 dr to 1½ oz water) instead of avertin. For a sudden severe respiratory spasm chloroform may be required if the patient is not yet under the influence of avertin.

*General treatment*—The patient should be kept isolated in a quiet darkened room, and his bedclothes should be cradled. He should be given feeds of glucose lemonade and milk alternately every hour. As thirst is a prominent symptom fluids are taken eagerly. Swallowing is made more easy by avertin, as it relaxes the trismus. If necessary, however, a nasal tube should be passed when the patient is under avertin, after which continuous feeding by the drip-method can be used. All manipulations should be done during the two to six hours following a dose of avertin, when the patient is most deeply anaesthetised.

#### LOCALISED TETANUS

Until the war of 1914-18 the only form of local tetanus which had been recognised was cephalic or head tetanus. A few cases had been recorded, in which the spasms began in the injured limb and remained most severe in it after generalisation occurred, but the first case of tetanus, in which the symptoms were strictly localised to one limb throughout the illness, was described in 1915.

by Courtellemant. Numerous observations on the subject were subsequently published in France and England but even at the end of the war many cases of localised tetanus probably escaped recognition through want of familiarity with the condition. Although there is no doubt that the extensive use of prophylactic injections of antitetanic serum was the cause of the relative frequency of localised tetanus since 1915 the disease was not an entirely new one, but existed before the introduction of serum prophylaxis, as at least one typical case was observed which occurred in spite of the fact that no serum had previously been injected (Claude and Lhermitte, 1916)

The large majority of cases of localised tetanus occur as a result of the protection afforded by prophylactic injections of serum being sufficient to prevent generalization, but insufficient to prevent the production of symptoms by the action of the toxin, which is absorbed by motor nerve endings near the wound and acts upon the corresponding segment of the spinal cord.

#### Symptoms

(a) Cephalic or head tetanus.—In 1867 Rose described a form of tetanus, in which the spasms were confined to the masseters and facial muscles, with the addition in some cases of the pharynx. This condition, which has been called cephalic or head tetanus is generally complicated by facial paralysis, and is always the result of a wound of the face or head, especially in the neighbourhood of the eye. The facial paralysis is generally confined to the injured side, but is occasionally bilateral. It may begin before any spasms occur but more frequently it develops at the same time as the trismus or shortly afterwards. It is never sufficiently severe to prevent spasms occurring at intervals in the parietal muscles, the cells in the medullary centre of the facial nerve being abnormally excitable owing to the action of the tetanus toxin. In most cases the paralyzing effect of the toxin disappears by the time the spasms have completely relaxed.

Cephalic tetanus was extremely rare in the last war less than half a dozen cases having been recorded.

(b) Tetanus localised to a limb.—The incubation period in some cases is as short as in ordinary tetanus, periods between five and fifteen days having often been recorded. Just as frequently the symptoms appeared between the twentieth and thirtieth days, and occasionally as late as the eight and ninth week. But the true

incubation period in most of the latter cases was really shorter, as the symptoms generally followed a few days after an operation, which must have disturbed the wound in such a way that a quantity of tetanus toxin was suddenly absorbed.

The premonitory symptoms, which I have already described as often occurring in protected individuals before the onset of generalised tetanus, may also precede the appearance of localised tetanus. The first obvious symptom is either twitching or stiffness in the muscles near the wound. The spasms consist either of painful clonic movements or of momentary and painless twitches. They become gradually less frequent as the stiffness of the limb becomes more pronounced, and in many cases they disappear entirely owing to the permanent tonic contraction of the affected muscles being the maximum possible contraction, so that additional twitching is impossible.

The contractions may remain localised to a single segment of the limb, but more commonly the whole arm or leg is involved. The tonic contraction produces a characteristic attitude. The leg is generally in a position of extension with the foot dorsiflexed, the limb being as rigid as a poker, less frequently the knee is flexed. The arm is generally flexed at the elbow, the upper arm is adducted and the shoulder shrugged.

In true localised tetanus the contractions remain strictly localised throughout. The nature of such cases might have been regarded as doubtful, were it not for the fact that every intermediate type was observed between the entirely local form and the severe generalised form beginning with slight local symptoms. In the intermediate cases the indications of generalisation may be very slight and last only twenty-four hours. There may be slight difficulty in swallowing or some pain and stiffness is felt in the masseters or neck, especially at the height of spasms affecting the limb. But neither definite trismus nor retraction of the head is present, and the chest can still be touched with the chin. The muscles of the face and trunk are not affected. These slight indications of generalisation must be distinguished from the result of direct spread, in which the muscles of the trunk in the neighbourhood of the affected limb are involved. Thus spasm of the lower abdominal muscles of the same side may be present in local tetanus of the leg, and the scapular and pectoral muscles were contracted in two of my cases of tetanus of the upper arm. Moreover, the

spasms occasionally spread to the corresponding limb of the opposite side, though they are never as powerful as on the wounded side this is very rare in the case of the arm, but several cases of so-called paraplegic tetanus, in which both legs are affected have been described.

Uncomplicated localised tetanus is probably never fatal, as there is no danger of asphyxia or respiratory complications, and the general toxæmia is comparatively slight.

### Diagnosis

Most cases of localised tetanus which occurred before the middle of 1915 must have been diagnosed as traumatic contractures, and five out of the nine cases I saw during the last two years of the war were at first regarded as hysterical in origin. On the other hand the contractures in at least two cases which have been published as examples of local tetanus must really have been hysterical as the onset was immediately after the wound although it is impossible for tetanus to develop until the bacilli introduced into the wound have had time to produce toxin which has then to travel to the central nervous system before it can give rise to symptoms.

Spasms developing immediately after a wound is inflicted are generally reflex and protective in nature but they often continue as hysterical symptoms after the first few minutes or hours they are never caused by tetanus. A later onset is compatible with hysteria or local tetanus, and in both the extent of the contracture is often out of proportion with the size of the wound. If the contracture persists in sleep, hysteria can be excluded. A general anæsthetic causes hysterical contractures to disappear as soon as consciousness is lost but tetanic contractures persist, and even under deep anæsthesia they may be still present in a minor degree. If the muscles are of a wooden and unvarying hardness, tetanus is almost certainly present. An increase in the size of the muscle, possibly due to obstruction of its lymphatics, without tenderness or subcutaneous œdema, is conclusive evidence in favour of local tetanus.

When there is any doubt as to the diagnosis the condition should be assumed to be local tetanus, as in local tetanus it is never possible to say whether generalisation may not develop later. Early injections of antitetanic serum should prevent this, and they will do no harm even if the spasms are not really due to tetanus. When

ever it is possible that the spasms are wholly or in part hysterical in origin, the effect of psychotherapy should be tried

*Localised tetanus following a wound of the pectoral muscles*—Pte M, aged 31, was wounded on July 25th, 1916, by a small fragment of shrapnel, which lodged in the outer part of the left pectoralis major without injuring the ribs. Nineteen hours later he received a prophylactic injection of antitetanic serum. Two small pieces of metal were removed from the wound on August 17th. The next day he had a "jumping" sensation over the wound, which was now about the size

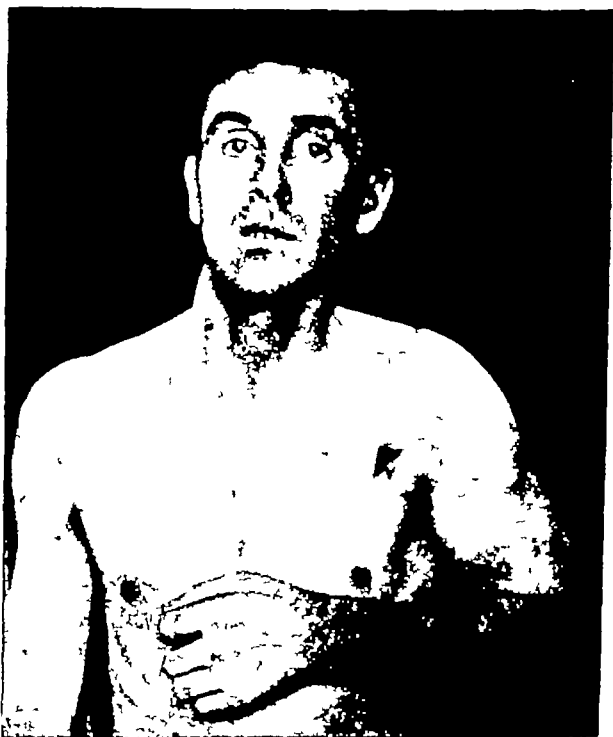


FIG 43—Case of tetanus, localised to left arm, left side of neck and pectoral muscles, following a wound in outer part of pectoralis major

of a shilling. On August 20th he complained of spasmodic pain in the left arm, which soon became so severe that it could only be relieved by injections of gr  $\frac{1}{4}$  of morphia. When he came under my care on the 30th, the pain had much diminished, the outer part of the pectoralis major, though not the clavicular portion, together with the biceps, brachialis anticus, deltoid, trapezius, and supra- and infraspinatus were in a condition of constant spasm (Fig 43). Some days later the triceps and flexor carpi radialis also became affected. The patient's head was slightly inclined towards his left shoulder, which was considerably raised, his arm was at first slightly abducted, but later it

was held firmly to his side and the forearm was fully flexed the hand was semi-pronated the palm being in contact with the epigastrium. The muscles of the forearm and hand were not affected. The spasm was so extreme that it could not be overcome even sufficiently for a towel to be inserted in the flexures. The affected muscles were of a wooden hardness. The circumference of the left arm was 13 inches that of the right, when the biceps was contracted as strongly as possible was only  $11\frac{1}{2}$  inches, although the patient was right-handed. The increased size of the muscles was so great that the skin could hardly be raised from them, but there was no oedema, and the forearms were equal in circumference. The contracted muscles were not tender on pressure. No sound could be heard on auscultation of the muscles there was no variation in the degree of contraction, and no twitching occurred on touching the skin or with excitement. There was no rigidity of the muscles of the jaw neck, abdomen or right arm. The constant spasm produced an aching pain which interfered with sleep. Major W J Turrell applied diathermy to the muscles, which relaxed slightly so that they could be tested electrically the reactions to both faradism and galvanism were normal and equal on both sides. A hot bath also produced slight temporary relaxation. Relaxation was very incomplete even under deep general anaesthesia. The skin over the contracted muscles felt warmer than on the other side and the patient himself noticed the difference, but otherwise sensation was unaffected.

On August 23rd and September 7th and 11th 1 000 units of anti tetanic serum were injected subcutaneously. The same quantity was given intrathecally on September 9th. The cerebrospinal fluid was under normal pressure. It contained no cells and had no effect when injected by Professor Sherrington into the muscles and nerves of cats.

By September 11th the muscles except the pectorals, had considerably relaxed. The arm was now only painful at night. Its circumference was  $11\frac{1}{2}$  inches, and by September 16th it was only  $11\frac{1}{4}$ . On the 22nd the deltoid and the outer part of the pectoralis were the only contracted muscles.

From this time the patient's condition steadily improved but when I last saw him on November 30th the deformity caused by the contraction of the muscles had not completely disappeared and he was unable to raise his arm above his shoulders in spite of exercises and passive movements. The increased size of the muscles had given place to definite atrophy the circumference of the left arm being now only 10 inches. He felt very well and was able to do light work in the garden.

#### Treatment

Localised tetanus, if recognised early should be treated like ordinary tetanus, as it is impossible to foretell whether generalisation will occur. If recognised only at a later stage, when the danger of generalisation is almost negligible, it is sufficient to inject 20 000

units intravenously The stiffness, which often persists for weeks or months after the acute stage has passed, requires regular passive and active exercises, preferably after the application of radiant heat

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## CHAPTER XXVII

### WAR NEPHRITIS

War nephritis is a form of acute glomerular nephritis, which assumes an epidemic character among soldiers on active service. In the last war the condition was also called trench nephritis, but the name was unsuitable, as there were many cases in men of the Army Service Corps, ammunition columns, headquarters troops and R.A.M.C., who had never been in the trenches.

#### *Ætiology*

Acute nephritis is a comparatively rare disease among adults in civil life. It does not appear to have been common in any campaign before that of 1914-18 except the American Civil War in which over 14,000 soldiers of the Northern Armies were invalided for nephritis, and to a less extent in the Franco-Prussian War. The disease appeared in the spring of 1915 and its incidence steadily rose till June, by which time there had been 1 061 cases in the British Army in France and Flanders. After this the incidence remained fairly constant till the end, with irregular waves which did not depend upon the weather or temperature. The hospital admission rate in France for nephritis was 7·4 8·5 9·5 and 4·2 for 1 000 for 1915 1916 1917 and 1918 respectively. Very few cases occurred among French troops until July 1915 by which time the infection appears to have spread from the neighbouring British lines. There were similar but less severe, outbreaks among the troops at Gallipoli, except at Suvla, and at Salonica, and in the Italian Army in 1916. The disease was very prevalent among German and Austrian soldiers on every front after the spring of 1915 but not a single case was observed in the Turkish Army. It was rare among officers of all nationalities but was less so at the end than in the first year of the war. No officers died in France from nephritis in 1915 and only two during 1916. No cases of genuine war nephritis occurred in training camps in England.

The majority of cases occurred among soldiers who were previously healthy. There is no doubt that an earlier attack of acute nephritis rendered an individual more liable to develop the disease, which then tended to be unusually severe. In a series of 571 cases of war nephritis 11 per cent. gave a history of a former attack (Bradford,

units intravenously The stiffness, which often persists for weeks or months after the acute stage has passed, requires regular passive and active exercises, preferably after the application of radiant heat

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few cases *B. coli* was isolated, but this was probably due to coincident intestinal disturbances and was of no pathological significance. Bronchitis preceded or accompanied the nephritis in 30 per cent. of the cases (Hume) it was, however common among Indian troops in spite of their freedom from nephritis.

There is some evidence that the disease is contagious. A sister and two orderlies, who were on duty in the nephritis wards of a general hospital, contracted the disease, though they had never been to the front, and the laboratory attendant, who examined all the urines of nephritis cases, also developed the disease (Davies and Weldon, 1917). The contagion was not present in the soil, as a division already providing a moderate number of cases continued to do so when moved to a different part of the line and another division, which had hitherto been comparatively immune, became no more affected than before when it took over the trenches belonging to troops among whom the disease was well established (Dunn and McNee, 1917). There is, however something about the conditions of life of the soldier as distinct from the civilian, which makes him specially liable to acute nephritis, as it did not occur among the civilian population or refugees of Belgium or France living among soldiers, many of whom were billeted on civilians. This was in striking contrast to the parallel incidence of most epidemic diseases among civilians and soldiers.

The incidence of the disease was never sufficiently great to be of any real importance to the strength of the Army. In the records examined by Dunn and McNee the largest numbers occurring in a battalion were ten cases in four months. Other battalions in the same division had three or four or perhaps not a single case in the same period. Sometimes four or five cases occurred in the course of a few days in a unit which had been free from nephritis for several months, and the outbreak was not necessarily followed by the occurrence of additional cases.

### Morbid Anatomy

Dunn and McNee had unique opportunities of investigating the morbid anatomy of trench nephritis, as they examined the organs of thirty five cases, in which death occurred between the second and fourteenth day and in which there was no evidence, such as overgrowth of connective tissue, pointing to pre-existing renal disease. The pathological features were remarkably uniform. The appearance of the kidneys to the naked eye was almost normal. A

1922) In a few cases with no definite history the symptoms or the autopsy in the acute stage pointed to the existence of chronic nephritis

Exposure to cold or wet aggravates war nephritis when it has once developed and may produce an acute attack in men whose kidneys are already damaged, but it was certainly not an important factor in the large majority of cases. The disease was rare in France during the first winter of the war in spite of the frequency of exposure to cold and wet, it became common in March and April 1915, when more cases occurred than in the preceding seven months, and the high incidence continued through the summer. The majority of cases before March 1915 were acute exacerbations in middle-aged men with chronic renal disease and were not genuine examples of war nephritis. Sudden changes in temperature also appear to be of small importance, as such changes were never as extreme on the western front as in the South African and Russo-Japanese wars, in both of which nephritis was very rare. If cold had been an important factor, Indian troops would probably have suffered greatly from the disease, but only three cases were reported among them, this was perhaps due in part to the protection against chills afforded by the loin cloths they constantly wore.

The nephritis might conceivably have been due to metallic poisoning caused by the consumption of tinned rations, but no trace of metallic poison was found in the urine. War nephritis is certainly not a result of the chlorination of water, as it was just as common among men who never had any as among those who constantly drank it. Alcohol was not a factor in its production, 13 out of Hogarth's 75 cases were total abstainers.

Acute tonsillitis and other infections may be followed by nephritis among soldiers as among civilians, but this accounted only for a very small proportion of the cases. Cultures from the throat in ten soldiers with nephritis did not differ from cultures from ten wounded soldiers, and in both cases the throat was less septic than in ten civilian patients who were in the same hospital for various surgical conditions (Cantlie). A positive Wassermann reaction was found no more frequently than in the army generally.

Diarrhoea frequently preceded the onset of nephritis in Gallipoli, but this was probably accidental and due to the extreme frequency of the former, as no such history was obtained in the majority of cases occurring elsewhere. The urine was generally sterile, in a

been gradually replaced to a large extent by hæmaturia as the most obvious symptom in both the idiopathic and scarlatinal nephritis of civil life. He suggests that its frequency as a premonitory symptom in war nephritis was due to the adverse conditions under which it was contracted, as a state of subnormal health apart from kidney disease is accompanied by a tendency to œdema. Slight ascites sometimes occurred, but hydrothorax was rare. Pulmonary œdema was common.

The headache, more commonly frontal than occipital, was often sufficiently severe to prevent sleep. It was sometimes accompanied by vertigo. The breathlessness was a very constant and characteristic early symptom. At the onset it might be present only on exertion, but it generally became continuous by the time the patient reached hospital, when it was apt to be most marked at night. It was associated with short and rapid respiration and was sometimes so urgent and distressing that it suggested the presence of acute bronchitis or pneumonia. It was primarily due to pulmonary œdema, but bronchitis was a contributory factor.

There was often some pain in the lower part of the back and the loins. I frequently noticed tenderness in both loins, and in some cases the kidneys were palpable and appeared to be enlarged as well as tender. The pain was probably due to stretching of the elastic capsule of the congested kidneys. The temperature was often raised to 100° or 101° at the onset, but numerous cases of equal severity were completely afebrile from the earliest stages. Sweating was much more frequent than in other forms of nephritis, and *herpes labialis* was sometimes observed. Bronchitis was often present and might cause a troublesome cough. In cold weather severe bronchitis and broncho-pneumonia were common, but pleurisy was rare, and pericarditis was never recorded.

Nausea, vomiting, slight drowsiness and apathy were frequently present. Epileptiform convulsions sometimes occurred suddenly in apparently slight cases, generally early in the course of the illness but the blood pressure was then always high. Severe *uremic* convulsions, if not fatal within the first few hours, were generally followed after a few days of semi-coma by rapid recovery (Sundell and Nankivell, 1917). An ammoniacal odour was often noticeable. Amaurosis might occur and I saw one case with transitory acute mania. Cutaneous eruptions were rare, and cramps and hiccough dyapnoea did not occur. The terminal symptoms in cases dying

few were slightly enlarged, and the cortex was often pale and the pyramids somewhat congested. The capsule was never adherent and the surface was smooth. The only constant change occurred in the capillary blood-vessels of all the glomeruli uniformly through the whole kidney. There was often some hyaline thickening of their walls, the capillaries were dilated and more or less completely obstructed by numerous cells, consisting chiefly of swollen endothelial cells with a few polymorphonuclear leucocytes and lymphocytes, but few or no red corpuscles. The vascular obstruction gave rise to secondary fatty degeneration and catarrh of the glomerular epithelium and to a less extent of the epithelium of the tubules. There was no evidence of inflammatory reaction, no cellular or fibrinous infiltration of the connective tissue being visible, even round the glomeruli, but small hæmorrhages were occasionally found. Beyond the slight secondary changes already mentioned, the tubular epithelium was remarkably little affected. Some long hyaline casts and small collections of blood were found in the tubules.

In all but two out of the twenty-two cases, in which Dunn and McNee examined the lungs, pathological changes were present. In sixteen characteristic alterations were found, in addition to the œdema which was constantly present and the acute bronchitis or broncho-pneumonia which was one of the chief causes of death. The walls of the terminal bronchi with their infundibula and occasionally the mucous membrane of the bronchioles and some of the larger bronchi were swollen with œdema and the lining epithelium was lost, the surface being covered by fibrin. Many of the corresponding pulmonary capillaries were thrombosed, but no inflammatory reaction was present. There was no exudation into the alveoli in the absence of broncho-pneumonia, and, when this was present, other parts of the lungs generally showed the characteristic changes just described, which had only been observed before as a result of the inhalation of irritant gas.

### Symptoms.

The first symptoms were generally œdema, headache and breathlessness. Œdema, though uncommon in the cases which occurred before March 1915, was later almost always present. It was rarely universal and never severe. It began in the eyelids, face, feet and hands, and after a short time it involved the back and flanks. Osman has pointed out that in the last seventy years œdema has

epithelial blood casts were uncommon. A varying and occasionally considerable amount of blood was present fever was generally well marked and oedema slight or absent in cases in which the urine was at first smoky or red. There was often a good deal of pus consisting of polymorphonuclear cells and large round mononuclear cells, often containing blood pigment this was probably of renal origin, as definite evidence of pyelitis was rarely present.

Improvement occurred rapidly. When fever was present the temperature generally fell in a few days, but occasionally remained slightly raised for some weeks. Sometimes the oedema lasted only two or three days it generally disappeared within ten days and was rarely present longer than a fortnight by this time the patient often felt quite well. Uremic symptoms disappeared within the same period. The blood pressure fell the oliguria was replaced by polyuria, and the abnormal constituents of the urine diminished in quantity disappearing completely in three or four weeks in most cases, although a little albumin, a few casts, and occasionally a good deal of blood might remain for a considerable period after all other symptoms had disappeared. Sometimes the albuminuria was intermittent before it finally disappeared. Occasionally blood reappeared in the urine and the albumin increased in quantity other symptoms might return, but a recurrence of oedema was rare. A return of albuminuria and sometimes of blood but without other symptoms, sometimes occurred after they had been absent for some weeks, generally as a result of an indiscretion in diet or over-exertion.

A reduction in haemoglobin out of proportion to that of red corpuscles occurred in the more chronic cases, and there was generally a relative increase of large mononuclear cells in the blood.

It must be remembered that no simple methods of measuring blood urea were available during the war period, so that very few estimates were made, but Giroux and Quirin (1916) found that the blood contained excess of urea during the acute state the percentage then gradually fell to normal, except when there was evidence of preceding chronic nephritis.

#### Diagnosis

Before labelling a case of nephritis in a soldier on active service as war nephritis, it is necessary to exclude the ordinary causes of nephritis, such as scarlet fever acute tonsillitis and jaundice. In

from uræmia were oliguria, headache, vomiting, rapid and laboured breathing, muscular twitching, occasionally general convulsions, and finally coma.

The blood pressure was always found to be raised if taken in the evening. Those observers who found the rise less constant probably made their records in the morning, as there was a considerable diurnal variation, the pressure at 6 p.m. being often 20 to 60 mm higher than at 10 a.m. (Abercrombie, 1918). An evening rise of blood pressure is, however, common in all forms of Bright's disease in which the arterial pressure is raised at all (Osman). The increased severity of the headache and dyspnoea at night was probably connected with the rise in blood pressure. The increased pressure often lasted only from five to ten days, but in some cases it continued for a few weeks. The morning systolic pressure was generally between 135 and 180 mm, it was highest when uræmic symptoms, especially convulsions, were prominent, and it might then be even 200 mm. The heart was not dilated, and, as in other forms of acute nephritis, cardiac hypertrophy could never be demonstrated earlier than six weeks from the onset, and even then it was rare and very slight. True albuminuric retinitis occurred only in rare cases with persistent albuminuria and a high blood pressure, when the disease appeared to be becoming chronic. Slight hyperæmia and œdema of the optic disc were, however, not uncommon in the early stages, and several cases of transitory retinœdema occurred, sometimes with plaques and occasionally detachment of the retina, but very rarely hæmorrhages or changes in the blood-vessels (Jessop). The retinal changes disappeared about the same time as the facial œdema.

The urine was often scanty at first, especially when there was much œdema. Uræmic symptoms were sometimes accompanied by suppression for twelve or twenty-four hours, but in many cases there was little or no reduction in the quantity, and Dunn and McNee recorded the case of a man dying from uræmia, who passed 52 oz. of highly albuminous urine in twelve hours. The quantity of albumin varied greatly in different cases and from day to day in the same case, it was often considerable at first. The amount frequently diminished greatly in the first few days, and it might almost disappear in a week. Casts were generally found in large numbers, the majority were hyaline, many containing a few intact cells from the renal tubules, others were partly granular and partly

It is important to distinguish war nephritis from acute exacerbations of ordinary chronic renal disease. The latter occurs as a rule in older men, the heart is hypertrophied, the blood vessels thickened, retinal changes are present, and cedema may be slight or absent.

### Prognosis

The immediate mortality of war nephritis was about 3 per cent. Death was almost always due to uræmia, acute bronchitis or bronchopneumonia. About 65 per cent. recovered completely generally within four weeks, and returned to duty showing no further evidence of nephritis. The remaining 35 per cent. were invalided from the service for nephritis. Of these about 56 per cent. recovered completely mostly within a year of the onset, but a few up to five years, and a small number between six and ten years from the onset (Osman, 1936). The mortality of those who were discharged from the army on account of nephritis can be estimated from the fact that out of 13,550 men receiving pensions for nephritis 4 118, or 30 per cent. had died by 1927, probably two-thirds from chronic nephritis and the remainder from other causes.

Further details about the prognosis can be gathered from the detailed investigations of Abercrombie on a small series and of Osman on a very large series of cases.

Abercrombie investigated the after history up to November 1917 of 171 unselected cases, which had been under his care at a base hospital in France between April 1915 and February 1916. Six had died, one in France, three in English hospitals and two after discharge from hospital. Of the remaining 165 32 were invalided for nephritis during their first period of treatment in English hospitals, and 22 more were subsequently discharged for nephritis after returning to some form of duty 109 (65 per cent.) returned to duty and showed no further evidence of nephritis 79 of these became first-line troops. Of 68 cases, in which the period from the onset to the disappearance of cedema and uræmic symptoms and the re-establishment of the urinary flow was more than 24 days, 50 per cent. died or were invalided, whereas of 97 in which this period was 24 days or less, only 27 per cent. died or were invalided. Four patients subsequently developed pulmonary tuberculosis.

After the war the Ministry of Pensions arranged that soldiers

France and Flanders when jaundice was present, however slight, nephritis was generally a symptom of infection with *Leptospira ictero-haemorrhagica*, and in Gallipoli and Mesopotamia of epidemic catarrhal jaundice. This corresponds with the fact that jaundice in civil life is one of the commonest causes of acute nephritis apart from scarlet fever and tonsillitis. In the Austrian army many soldiers took cantharides and chromic acid in order to gain their discharge on account of nephritis, but no case of self-produced nephritis was recorded in the allied armies.

Young soldiers are occasionally sent from their units to hospital as cases of nephritis, but on examination of the urine no albumen is found. These are probably cases of functional albuminuria and not nephritis at all. A man may have fallen out during a march on account of faintness, and examination of his urine has shown the presence of albumen. This is simply due to excessive exertion, and is comparable to the temporary albuminuria which frequently occurs after athletic contests. These patients can be sent back to their units after a short rest, as the albumen generally disappears in two or three days and almost always within a fortnight, and this form of albuminuria does not in any way predispose to nephritis. Lyall (1941) found albuminuria on more than one occasion in 110 out of 20,000 men (0.55 per cent.) when examined for recruitment to the Services. Of these 31, or about one-third, were cases of functional and transient albuminuria: the urine passed during rest was free from albumen, it contained no casts, red corpuscles or leucocytes, the blood urea and the urea concentration test were normal, there was no recent history of an illness which might cause nephritis, and there was no rise of blood pressure. In an additional 22 some feature was present, such as albumen at rest, occasional granular or hyaline casts, red corpuscles or pus cells in the urine, or a rise of blood pressure, which pointed to possible slight nephritis. In 14 there was more definite clinical or pathological evidence of recent subacute nephritis and in 31 of chronic nephritis, although the men felt well and had not sought medical advice. Finally 12 showed evidence of urinary infection, including tuberculous. McLeod and Ameulle (1916) found that the incidence of albuminuria in apparently healthy British and French troops was between 1.6 and 4.7 per cent. under various conditions in France, but was as high as 10.1 per cent. among recent recruits during strenuous training in England.

Immediate mortality (uræmia broncho-pneumonia)		3
Complete recovery and return to duty		64
Invalided		33
recovery mostly within a year but up to 10 years		18
chronic albuminuria with no signs of nephritis		2
chronic nephritis without hypertension		3
chronic nephritis with hypertension		3
died of uræmia		7
		<hr/> 100
Total deaths	10	
Total recovery	82	
Chr. albuminuria	8	{ Of these some may have later recovered and some died of nephritis.
Chr. nephritis		

These figures are very similar to those recorded by Ellis (1942) in his analysis of 225 cases of nephritis among civilians under his care at the London Hospital 4 per cent. died in the acute stage 4 per cent. ran a rapidly progressive course ending in death within a few months, 10 per cent. apparently became chronic and 82 per cent. made a complete recovery

### Is War Nephritis a Specific Disease ?

The epidemic character of the disease is the most striking feature of war nephritis. Its clinical features are essentially similar to those of acute nephritis in young adults in civil life. Oedema is a more common and hæmaturia a less common presenting symptom. The frequency of dyspnoea, the rarity of inflammatory complications, and the low immediate mortality in spite of the liability to sudden attacks of sudden uræmic convulsions are other striking features of war nephritis. The late results of acute nephritis are approximately the same in adult civilians as in soldiers on active service (Osman)

The morbid anatomy of war nephritis does not differ in any way from that of acute nephritis in civil life. It seems probable that it is not a specific disease, but is a form of nephritis occurring among soldiers and presenting certain unusual features as a result of the very different conditions of life of a war worn soldier and a civilian of the same age.

### Treatment

Rest in bed and warmth are the first essentials in treatment. This should be continued until all symptoms have completely disappeared and, if possible, until the urine is normal. If, however after an initial period of two months in bed the condition of the

receiving pensions on account of war nephritis should be examined annually by MacLean and de Wesselow. The records of the observations they carried out between 1920 and 1926 on 5,120 cases, in which the data were sufficiently detailed, were analysed in 1936 by Osman. He found that they fell into six groups

(1) *Complete recovery*—In 37 per cent there was finally no albuminuria or other sign of kidney disease. In some albumen was already absent at the first attendance, but in others it gradually disappeared during the following years, even as long as ten years after the onset.

(2) *Recovery except for slight albuminuria*—In 19 per cent the albumen steadily diminished from a previously larger amount, often at first accompanied by casts or red cells. As many cases in group (1) passed through this stage before ultimate recovery, it can be concluded that the large majority of the cases in group (2) can be added to those of group (1), making a total of 56 per cent with complete recovery.

(3) In 11 per cent *well-marked albuminuria* persisted without other signs of nephritis.

(4) *Chronic nephritis without hypertension*—In 15 per cent casts as well as albumen were present. The albumen varied from a trace to a large quantity and was sometimes accompanied by red corpuscles, slight oedema of the feet and legs, and occasionally by nitrogen retention. Some of these probably recovered completely and others drifted into the remaining classes.

(5) *Chronic glomerular nephritis*—In 12 per cent classical signs of chronic glomerular nephritis were present.

(6) *Persistent hypertension*—In 5.5 per cent hypertension was the only abnormal sign, except for a trace of albumen without casts in two-thirds of the cases.

Subsequent attacks occurred in 7 per cent of the whole series of cases, but this did not appear to prejudice final recovery.

The following table shows the fate of a hundred army cases of war nephritis in the course of ten years as calculated from the above statistics.

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urine shows no further improvement during a third month, the patient can be allowed to get up in spite of the persistence of albuminuria. A return of symptoms after getting up is, however, an indication for a further period in bed. The diet should consist for the first two or three days of glucose lemonade and fruit. Farinaceous puddings, milk, bread and butter and vegetables can then be added. A larger quantity of nitrogenous food should be allowed only when any headache and dyspnoea which may have been present have disappeared; an ordinary diet can then be given whatever the condition of the urine. In the rare cases in which the oedema shows a tendency to persist a salt-free diet generally leads to its rapid disappearance. The bowels should be kept regular by paraffin and, if necessary, plain water enemata.

Severe headache is relieved instantaneously by lumbar puncture if the pressure of the cerebro-spinal fluid is high. Uræmic convulsions call for immediate removal of 20 oz. of blood, and their return can be prevented by lumbar puncture.

Great care should be taken in the transport of nephritis patients, as the fatigue and exposure likely to occur in long journeys may lead to aggravation of the symptoms.

Owing to the danger of relapse on exposure to the hardships inseparable from active service, few men who have once suffered from acute nephritis can safely return to more than light duty at home, and then only if there is no albumen when they are on full diet and doing a moderate amount of work. An exception can be made for very slight cases, which appear to recover completely within two or three weeks, but only after a preliminary period of three months' light duty followed by three months' full duty at home. During the whole of this period the soldier should be under medical observation, and his urine should be examined once a week.

If the albuminuria persists for more than six months, the patient should be discharged from the army, but as much as nine months may be allowed for young men in whom no cardiovascular changes have developed. There is, however, no reason why such men should not be employed as clerks or at other indoor sedentary occupations with the army at home. But no patient can be regarded as cured until albumen has been absent for three months in spite of a full diet and an average amount of muscular exertion.

That pyogenic infection of the skin (pyoderma) was the preponderating cause of skin disease on the western front is illustrated by the following figures collected by Semon and myself (1917). Out of a total of 669 cases observed by us and admitted to hospital between April 1st and May 9th, 1917 631 were suffering from pyoderma. Of these in 452, or 71·6 per cent., the pyoderma was secondary to parasitic infection. Scabies alone was responsible in 171 cases (37·6 per cent.), *Pediculus corporis* in 257 (56·9 per cent.) while in 24 (5·5 per cent.) the two infections co-existed. In 112 of the remaining 179 cases of non parasitic pyoderma the pyogenic infection appeared to be a complication of the seborrhoeic state and in 67 no definite predisposing cause could be determined. It should be noted that out of the total of 669 cases only 38 (5·6 per cent.) were suffering from diseases other than pyoderma, e.g. psoriasis, herpes zoster eczema, ringworm, etc.\*

## SCABIES

### Ætiology

For contagion with the acarus of scabies to occur it is probably essential that one or more impregnated young females which are actively mobile at night-time, should be transmitted from an infected person. This may result directly and almost inevitably from sharing the bed of a scabietic, thus accounting for familial epidemics and for the frequency of a venereal origin of the disease. Mellanby (1941) with the aid of volunteers investigated the possibility of transmission of scabies by contact with bed clothing and underclothing. Contrary to what has hitherto been believed this would appear to be a rare cause of contagion. In 63 experiments with underclothing and blankets the disease was transmitted only twice, although everything was done to favour transmission. In none of the 25 experiments with blankets alone did infection occur. On the other hand, transmission by comparatively slight personal contact is readily accomplished. Mellanby concludes that scabies is usually transmitted by personal contact either of a slight or of a venereal nature. He points out that small children are often the first members of a family to be infected. They probably then infect their mothers, who in turn transmit the disease to their husbands.

\* For a full statistical study of the incidence of scabies and pediculosis in the last war vide Volume 2 of the *Official History of the War: Medical Services* (London, 1923).

## CHAPTER XXVIII

### SKIN DISEASE IN WAR

BY H. W. BARBER, F R C P

#### Introduction.

The following account of diseases of the skin in the last war is based, apart from a study of the literature, on my experience over a period of several months in 1917 at a base hospital in France, which was largely devoted to dermatological casualties. No attempt will be made to deal with diseases which occurred in the Eastern or other theatres of war.

As will be evident later, the enormous number of casualties from skin diseases on the Belgian and French fronts was primarily due to the prevalence of two animal parasites—the *Pediculus corporis* and the *Acarus scabiei*, and secondarily to the severity of the superadded pyogenic infection which these engendered. That such infection should occur was inevitable in view of the conditions of trench warfare that then existed, but apart from trauma of the skin by scratching and the restricted opportunities of washing and changing clothes, there was another very important factor which unquestionably favoured the growth of pyogenic organisms in the skin, namely the seborrhœic state. This in turn was favoured by the character of the diet that obtained among troops on active service.

We thus see that infestation by two common parasites, lack of cleanliness, and a defective dietary were the factors largely responsible for the predominant features of the dermatology of the last war. It is likely that the same factors accounted also for the large number of casualties from skin diseases that occurred in previous wars. For example, out of an army of some 600,000 men in the American Civil War 32,000 cases were diagnosed as scabies and 35,667 were merely recorded as "skin disease," and in the Napoleonic campaigns cases of scabies occurred in hundreds of thousands (MacCormac).

Under the improved conditions that our troops enjoyed in France up to the time of the evacuation in 1940 the incidence of skin diseases was very much less than in the war of 1914–1918.

between the severity of the eruption and the number of parasites, since those with few clinical symptoms may harbour large numbers and those with an extensive eruption and much secondary pyogenic infection very few. Pus appears to kill the parasites that come in contact with it.

In both scabies and pediculosis corporis the cutaneous lesions may be divided into primary ones caused directly by the respective parasites, and secondary ones resulting from scratching and super added infection with pyogenic organisms. In scabies those that may be regarded as *primary* are—

- (1) The burrow run, or cuniculus.
- (2) The vesicle.
- (3) The indurated papule.

The *secondary* lesions consist of—

- (1) Scratch marks.
- (2) Erythematous macules or patches and urticarial wheals or papules.
- (3) Various types due to secondary infection with staphylococci and streptococci e.g. pustules, which dry up to form crusts, boils, impetigo which may be of the bullous type, ecthyma and infective eczematoid dermatitis.
- (4) Eczema of non-infective origin, which occurs as a rule in those subject to eczema and probably results from scratching

**PRIMARY LESIONS** (1) *The burrow*—This is absolutely pathognomonic of the disease. It is formed by the impregnated female parasite in the deeper parts of the horny layer of the skin. Raising herself to an angle of  $45^{\circ}$  by means of her long posterior bristles, she tears through the horny layer until she has excavated a cavity, which she enlarges by lateral movements. She then by forward progression creates a tunnel, in which as she advances, she lays her eggs.

The fully formed burrow varies in length from two or three millimetres to a centimetre. Occasionally straight it is as a rule curved in the shape of an S or of a horse-shoe. It projects a little from the surface of the surrounding skin, so that it may be felt as a slight ridge on palpation. Very often greyish black points may be observed these are the openings in the roof through which freshly hatched larvae have emerged. The colour of the burrow depends to some extent on the habits of the host. Among cleanly persons it may be almost white and difficult to see but it can be

Among children scabies is doubtless spread during play by holding hands and putting their arms around each other's necks. Investigation of scabies in the Army shows that a large number of cases may be traced to infection on leave, and in many, as in the last war, a venereal origin is probable. There is little to suggest that the disease is spread from man to man in the Army in England, but I am satisfied that this occurred in the war of 1914-18 in France. Nurses may acquire the disease by tending scabietic patients confined to bed.

Although the acari, larvæ and nymphs can live only for a very short time away from the body, Munro (1919) showed that ova could be hatched out even when exposed to a temperature of 6° C for periods of from one to six days. But since they are normally hatched in the female burrow, it is not likely that they would often be found in clothing or blankets.

### Morphology of the parasites.

*The female*, just visible to the naked eye, is oval in shape, about one-seventieth of an inch in length, and greyish or reddish in colour. She has on the ventral surface eight legs arranged in pairs. The two front pairs are provided with suckers and the posterior ones with long bristles. *The male* is much smaller, reddish in colour, and very difficult to find. According to Munro (1919) he also makes small burrows at the time of mating. His hindmost legs have suckers instead of bristles. *The larvæ* are devoid of sexual organs, but, like the adults, the future males are smaller than the future females. They inhabit the burrows in which they are hatched or make new ones in the neighbouring hair-follicles. The nymphs, which represent the intermediate stage between the larvæ and adults, also appear to make small burrows. After about a month the sexually mature parasites are formed and copulation occurs. In a last moult at about the sixth week the ovipositor appears on the impregnated females.

### The Cutaneous Lesions of Scabies.

Johnson and Mellanby (1941) have investigated the numbers and location of adult female parasites in 886 men suffering from scabies. The average number per man was 11.5, 52 per cent harboured less than six parasites, and only 3.6 per cent more than fifty. In 63 per cent the mites were found on the hands and wrists, 10.9 per cent on the elbows, 9.2 per cent on the feet, 8.4 per cent on the genitals, and 4 per cent on the buttocks. There is no correlation

found and secondary lesions are scanty or absent. This papule is raised considerably above the level of the surrounding skin and is hard to the touch. In certain situations, e.g. the anterior axillary folds, it is elongated, in others, e.g. the scrotum it may be circular. Its sites of election are the wrists, elbows anterior axillary folds, the umbilicus, the scrotum and penis, and the buttocks over the ischial tuberosities. These indurated papules may persist and continue to itch for some time after the disease has been successfully treated.

**SECONDARY LESIONS.** (1) *Scratch marks*—One of the most striking features of the generalised scabietic eruption is the erection of the pilo-sebaceous follicles due to contraction of the pilomotor muscles. As a result of this the summits of the follicles are excoerated when the patient scratches, and consequently the majority of scratch marks consist of pin point blood-crusts at the apices of the erected follicles, thus contrasting with the linear excoriations of pediculosis corporis (Fig 44 Plate VII). These follicular scratch marks are as a rule well seen on the abdomen limbs and buttocks. At the same time some linear blood-crusts may also be observed.

It must be remembered that a similar follicular erection with pin-point excoriations occurs in certain cases of prurigo for example that of allergic subjects, in whom asthma and hay fever are so often coincident symptoms. In such cases, however diffuse lichenification and increased pigmentation of the skin always develop sooner or later and these are seldom seen to any degree in scabies. In this connection it should be noted that some eosinophilia may occur in scabies, but it is usually slight.

(2) In patients recently infected with scabies erythematous and urticarial lesions may be seen, but they are as a rule transitory and are later masked by the more characteristic eruption.

(3) Some degree of secondary pyogenic infection is almost always present in scabies except in early cases. Naturally it is more likely to occur in those who are indifferent to personal cleanliness, and when some active focus of infection, such as a boil, is already or has recently been present. It is apt to be severe in seborrhoeic subjects, in whose skin pyogenic organisms—particularly staphylococci—grow readily. The prevalence of the seborrhoeic state and the conditions under which our troops lived in the last war caused the majority of cases of scabies to be complicated by staphylococcal and streptococcal lesions of various kinds and usually of con

rendered more visible by touching it with ink or tincture of iodine, the resulting black or brown tint contrasting with that of the neighbouring skin. In others it entangles dust and dirt, which give it a greyish-black colour, but this may also be partly due to the animal's excrement, which is easily recognised intermingled with the ova under the microscope. At one end may be seen the point of entry, at the other is a slight eminence, beneath which the acarus lies and where she may be detected with a lens as a minute white point. By passing a needle through the roof of the burrow, it is possible after some practice to extract her for microscopical examination. It is simpler, however, to excise the whole burrow with a fine pair of scissors or a small scalpel, and to place it with the under surface upwards on a slide in a little dilute glycerine or *Liquor potassæ*.

Under a low power the curved outlines of the burrow can be recognised, and apart from the acarus itself there will be seen the ova in various stages of development, particles of excrement, and perhaps some newly-hatched larvæ. The ova situated farthest from the parasite are in the latest stages of incubation, and in them the structure of the enclosed larvæ may be studied. The burrow may be situated over a vesicle or pustule, but there is never any direct contact between their contents and the acarus.

Burrows are most likely to be found between the fingers, on the palms, the anterior surfaces and ulnar angles of the wrists, the ulnar borders of the hands, the elbows and anterior axillary folds, and the lower parts of the buttocks. In men the localisation on the prepuce and glans is of diagnostic importance, and here the burrow is likely to be associated with an cedematous papule often covered by a crust. Such a lesion may be of considerable size ("scabietic chancre"), and provide a portal of entry for the *Treponema pallidum* or of Ducrey's bacillus.

(2) *The vesicle*—In the majority of scabietic patients there are found, particularly on the lateral surfaces of the fingers and on the wrists, small vesicles with clear contents. They may be formed near or beneath the burrows or independently of them. From secondary pyogenic infection their contents may become purulent.

(3) *The indurated papule*—This is one of the most characteristic lesions of scabies, secondary in importance only to the burrow. Its formation is independent of the habits of the patient, and it may establish the diagnosis in cases in which burrows cannot be

complicate the secondary pyogenic infection that accompanies scabies would seem almost inevitable in some cases, for example in those with a predisposition to eczema, i.e. to sensitisation of the skin, and in the seborrhoeic state, which appeared to be the chief factor predisposing to its occurrence in the last war. It was this secondary eczematous infection of infective origin that rendered treatment of scabies more difficult and prolonged the convalescence for weeks or even months.

Although in these cases sensitisation of the skin to a staphylococcus was doubtless the usual if not invariable cause, it must be remembered that a similar sensitisation with consequent eczematous infection may occur to a streptococcus, and many cases of widespread, moist intertrigo are of this nature.

### Subjective Symptoms

After exposure to infection the burrows which are the pathognomonic sign of scabies, do not appear for some days. This period of incubation varies but is usually about ten days. It may be considerably shortened in hot weather and prolonged in winter. During it however the patient already becomes conscious of itching, which is usually at its worst during the early part of the night, and may be accompanied by outbreaks of erythematous and urticarial lesions. At this stage in the absence of burrows a certain diagnosis is difficult or impossible.

When the disease is fully established the itching is usually intense with nocturnal exacerbations, for it is the warmth of the bed that favours the activities of the female acarus. In those who work at night and sleep by day on the other hand, the itching is chiefly diurnal.

As with all potentially pruritic diseases the degree of itching varies greatly in different persons. While the nocturnal exacerbations are almost constant, some may complain of it being severe during the daytime. As might be expected, the more highly-strung and emotional the patient the greater the intensity of the subjective symptoms, and there is not necessarily any correlation between the severity of the eruption and that of the itching. Some, in whom large numbers of burrows are found with widespread secondary pyogenic infection, complain but little either of itching or insomnia. Very occasionally particularly in cases of long-standing, the pruritus may be almost absent.

*Acarophobia* is one of the forms of parasitophobia and is often of

siderable severity. These often retarded recovery, as they persisted or recurred long after the parasitic infection had been cured.

**Staphylococcal lesions.**—It has already been noted that the primary vesicular lesions of scabies may become secondarily infected, but apart from this staphylococcal pustules, which dry up to form crusts and are not necessarily follicular, are common. Such pustules are often seen on the hands, wrists and elbows. Boils and carbuncles also occur, and scabies may be the starting-point of a severe and intractable furunculosis.

**Streptococcal lesions.**—These consist of impetigo and ecthyma. The impetigo is often of the bullous type, the early lesions being bullæ with clear serous contents, which, however, rapidly become purulent from secondary staphylococcal infection, and then after rupture of the horny layer form the usual amber-coloured crusts. Under war conditions the streptococcal infection tended to spread deeply and to destroy the epidermis, thus producing ecthymatous ulcers covered with blood-stained and adherent scabs. Ecthyma, however, appeared to be a much commoner complication of pediculosis corporis than of scabies.

**Infective eczematoid dermatitis.**—By this term is meant an eruption having the clinical and histological features of eczema and due to infection with and sensitisation of the skin to a pyrogenic organism, as a rule a *Staphylococcus aureus*. It is very important to distinguish it from other forms of eczema of internal or external origin. Naturally it is a complication of some definite infective lesion of the skin or adjacent mucous membranes, such as furunculosis, a nasal discharge or otorrhœa, a chronic sinus leading to infected bone, or an infected wound. In such cases it begins around the primary site of infection and spreads peripherally; thence it may extend widely, and new patches may appear—often symmetrically—on distant parts of the skin. As might be expected, the eczematous vesicles are likely to become purulent, and independent pustules formed at the mouths of the pilo-sebaceous follicles are often seen. Wherever any part of the skin may be affected, the eczematization is apt to be most severe in the natural folds and flexures, where it takes the form of a moist, crusted and often malodorous intertrigo.

That a widespread eczematoid dermatitis of this type should

complicate the secondary pyogenic infection that accompanies scabies would seem almost inevitable in some cases, for example in those with a predisposition to eczema, i.e. to sensitisation of the skin, and in the seborrhoeic state, which appeared to be the chief factor predisposing to its occurrence in the last war. It was this secondary eczematisation of infective origin that rendered treatment of scabies more difficult and prolonged the convalescence for weeks or even months.

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*Acarophobia* is one of the forms of *parasitophobia* and is often of

serious import. It may occur in one who has had scabies and cannot be convinced that he is cured, or in those whose relations or friends have had the disease, or who have gleaned their knowledge of it from hearsay or reading. As with other sufferers of parasitophobia, the patient often severely damages his skin by rubbing in strong antiseptics or other irritants in a vain attempt to destroy the non-existent parasites. I met with several cases of acarophobia in the last war.

### Diagnosis.

It would be futile to discuss all the itchy dermatoses for which scabies might be mistaken. The important point is that the possibility of scabies should always be considered when a patient complains of widespread itching, no matter what his social class or habits may be. A knowledge of the distribution and the general features of the scabietic eruption, and a careful search for burrows and the indurated papules should prevent an error of diagnosis except in very early cases.

As pointed out by MacCormac and Small (1917), the clinical features of scabies in the last war differed in some respects from those presented by the average civilian case. Thus burrows and secondary lesions between the fingers were frequently absent, in sixty consecutive cases examined by Small they were present in only 13 per cent. But the elbows, anterior axillary folds, the umbilicus and abdominal wall, the penis and scrotum, and the buttocks over the ischial tuberosities were characteristically involved. Another important point was that the seborrhœic state predisposed to a widespread and severe infective eczematoid dermatitis, which tended to mask the underlying parasitic infection.

### Treatment.

The successful treatment of scabies is largely a question of technique. It is useless to tell the patient to rub in the ointment "where it itches," and he should be warned that if there is still irritation of the skin after the prescribed treatment has been thoroughly performed, it will almost certainly be due to the application employed and not to a persistence of the infection.

Important investigations upon the relative efficacy of several methods of treatment advocated for scabies have been carried out by Mellanby, Johnson, and Bartley, and will be the subject of a paper shortly to be published. They claim that by their technique over 90 per cent of ovigerous acari can be detected, and their

assessment of the success or failure of treatment is primarily based on an examination of the adult female parasites in treated cases. If only dead ones are found the method is presumably successful, but the results are checked by a study of the proportion of relapses. If, on the other hand living acari are found twenty four hours after completion of treatment the method is obviously a failure. The main conclusions Mellanby and his colleagues draw from their investigations are as follows.

(1) The classical treatment with *Ung Sulphuris* B.P (10 per cent.) is effective. A very high proportion of parasites are killed by one application and two applications cured 100 per cent of cases. The preliminary bath and scrub appear slightly to increase the efficacy of the treatment.

(2) *Ung Potassi Polysulphidi* (B.P.C) (Marcoussen's or Danish ointment, kathonan) proved to be even more successful, a single application curing all cases treated.

(3) Dusting the patient with flowers of sulphur is not only relatively ineffective even after three days treatment (67 per cent. of acari killed) but is likely to cause a severe dermatitis before a cure is ultimately achieved.

(4) Treatment with sodium thiosulphate and hydrochloric acid is equally unsatisfactory

(5) Even after three daily treatments with sulphur lather tablets about 50 per cent. of parasites were still alive.

(6) Derris root lotion and rotenone emulsion ( sarevan ) proved to be less effective than one application of sulphur ointment, and the emulsion (2 per cent. of rotenone) is apt to cause an intractable scrotal dermatitis

(7) Sulphur taken internally has no effect whatever on the acari, even after the ingestion of 10 grm. daily for ten days.

(8) Dimethyl-diphenylene-disulphide ( mitigal ) is perhaps the best of all applications. I used it almost exclusively for private patients before the war with apparently uniform success. Unfortunately it is now unobtainable. It is usually applied undiluted and then kills 100 per cent. of acari after one application. Mellanby's experiments suggest that even when diluted to 5 per cent. it is still effective.

(9) Benzyl benzoate which is the chief active ingredient of balsam of Peru, is lethal to acari both *in vitro* and when applied to the skin whereas sulphur ointment acts only in contact with the

body, since the parasites survive several days when placed in it on a glass slide. Owing to the shortage of animal and mineral fats, which makes the extensive use of ointments undesirable, it is now recommended that the standard treatment for scabies should be the careful application to the whole body from the neck downwards of the following emulsion of benzyl benzoate—

Benzyl benzoate	25 0
Lanette wax	2 0
Water to	100 0

This is more economical to use than the soap-spirit lotion, two fluid ounces of the emulsion being sufficient to cover the whole body as against  $3\frac{1}{2}$  oz of the lotion. Although one application of the emulsion is curative in a high proportion of cases, two are advisable either on successive days or at a week's interval. A preliminary bath and scrub are to be recommended, although they are not essential.

It is to be hoped that after the publication of these investigations the methods of treatment that have been shown to be ineffective will be abandoned.

The treatment of the disease in the war of 1914–18 differed in different centres. The routine method adopted at one base hospital was described by MacCormac and Small (1917), and consisted of—

(1) thorough lathering of the whole skin from the neck downwards with soft soap before and during a prolonged hot bath, and vigorous scrubbing with a brush in order to open up the burrows,

(2) the subsequentunction with *Ung Sulphuris* B.P. twice daily for three days,

(3) a second bath, after which the patient donned clean clothing;

(4) the application of a soothing cream or paste to counteract the irritant effect of the ointment,

(5) thorough disinfection of the clothing and bed-linen.

This method was cheap, easy to supervise, and suitable for dealing with a large number of cases, providing that an adequate number of baths was available. In my opinion, however, the *Ung Sulphuris* B.P. is unnecessarily strong and is apt to cause sulphur dermatitis of some severity.

The *Ung Potassii Polysulphidi* B.P.C., which is composed of polysulphides of potassium, equivalent to  $12\frac{1}{2}$  per cent of sulphur, with zinc oxide and benzaldehyde in a wool-fat and paraffin basis, is

extremely effective and much to be preferred to ordinary sulphur ointments. It is claimed that one application of this preparation suffices for cure, but in my opinion it is safer after the initial bath for at least three thorough inunctions to be given.

The method outlined above was found to be too drastic for cases with severe eczematization, most of whom were seborrheics, but the polysulphide ointments are usually well tolerated. A mild soap should be used in the initial bath, and instead of sulphur preparations storax ointment or one containing  $\beta$ -naphthol, balsam of Peru and storax, e.g.

R. $\beta$ -naphthols	5.0
Balsami Peruviani	15.0
Styracis Liq	20.0
Crete Præpt.	20.0
Adipis	40.0

may be employed.

The inunctions should be continued for a week.

In recent years treatment by a single application of a solution of benzyl benzoate after the usual preliminary bath has been popular in some clinics. Kismeyer (1937) claims that the method is quicker and less likely to cause dermatitis than treatment with polysulphide ointment (kathiolan) which he previously employed. The solution consists of equal parts of soft soap (B.P. 1932) isopropyl alcohol, and benzyl benzoate. About 150 gm. are required for each patient. The whole body is first rubbed with soft soap particular attention being paid to the sites of election. A hot bath is then taken, and in it the patient continues the rubbing for ten minutes. While still wet the body and limbs are then brushed over with the solution, the sites of election again receiving special attention. After being allowed to dry on, the solution is applied again for five minutes, after which the body is dried with a towel and the patient dons the clothing worn before treatment. Twenty four hours later another bath is taken and clean clothes put on. Underclothing is not disinfected, but washed and, when practicable, boiled. Bed-clothes should be disinfected or boiled.

Although one-day treatments with benzyl benzoate may be effective when carried out in hospital under supervision, I personally mistrust them when prescribed for ambulatory cases. As MacCormac (1941) points out, the ova of the acarus take three days to hatch, and the traditional treatment of seventy two hours with

sulphur ointment ensures that eventually insects only, not insects and ova, are being attacked Forman (1941), however, has found after three years' experience of treatment with benzyl benzoate in out-patient departments that it is as satisfactory as that with sulphur ointments.

The following is the routine method that I personally employ, and it has given me uniformly successful results.

(1) On the first night the patient should take a prolonged hot bath lasting about twenty minutes In the bath the skin of the whole body from the neck downwards should be thoroughly scrubbed with soap in order to open up all the burrows

After the bath the following ointment should be rubbed in thoroughly all over from the neck downwards Ung Potassii Polysulphidi (B P C), and the patient should get into old pyjamas, cotton socks and gloves

(2) On the next two mornings and two evenings further inunctions with the ointment should be made

(3) On the third morning a second bath should be taken, and the patient should put on entirely clean under-linen It is important that all used under- and bed-linen be disinfected or boiled

It is possible that the ointment may cause a sulphur dermatitis, and after the second bath a soothing cream may be applied

Zinci Oxidi	30
Lanolin Anhyd	50
Aq Dest	20
Vaselin Alb	200

Under no circumstances must the sulphur ointment be used for longer than the prescribed time, as, providing the treatment has been carried out thoroughly, any irritation that persists would be due to the sulphur and not to the scabies It is essential that all affected members of the household be treated simultaneously

As a rule after successful treatment of scabies the secondary pyogenic infection rapidly responds to mild antiseptic applications, but in cases with widespread infective eczematoid dermatitis convalescence may be prolonged for some weeks

### Prophylaxis.

As already pointed out, in civilian life scabies is almost invariably contracted either by sleeping with an infected person or through contaminated bed-linen, and the question arises how the spread of infection took place among troops on active service The probability is that blankets were the usual medium In many instances men were infected when on leave by prostitutes and conveyed the infection to their unit on their return In others

members of new drafts from England were doubtless responsible. Obviously efficient prophylaxis depended chiefly on thorough inspection of new drafts, men returning from leave and of all members of a unit in which cases of scabies had been discovered and disinfection of blankets that were suspect.

These measures, though excellent in theory were often difficult or impossible in practice.

## PEDICULOSIS CORPORIS

As Darier graphically put it, *les poux ont pullulé dans les tranchées pendant la guerre*, but even now the wastage of man power due directly and indirectly to lousiness is doubtless in sufficiently realised. It has already been pointed out that the body louse was responsible for more dermatological casualties in the war of 1914-18 than any other single cause and since the virus of trench fever and of epidemic typhus extensive outbreaks of which occurred in Russia and Germany and the spirochaete of one form of relapsing fever are conveyed by the parasite the total number of casualties due to it must have been enormous.

The extent to which our own troops were lousy had to be seen to be believed, and the kilts of the Scottish soldiers might have been specially designed for the benefit of the louse their pleats forming ideal shelters and breeding places for it. Peacock (1916) in his examination of 274 infantrymen of a division, which had spent six months in different parts of the line, found a lousiness averaging about 20 lice per man, but cases with a lousiness above 100 were not included. Nearly 5 per cent. of men were maximum cases. On them the number of lice found were 168, 180, 190, 376, 400, 552 and 895. Apart from these one shirt held 1,355 lice and 4,260 eggs, and another approximately 10,428 lice and 10,253 eggs.

Ninety five per cent. of men who had seen six months service in the line were found to be lousy and the striking difference between the average (20 per man) and the maximum cases raises the old question as to whether lice have a selective preference for certain hosts. In civilian life they appear to prefer the old and cachectic to children and healthy young adults, and Radcliffe Crocker records an interesting experiment by four young medical men. They placed a *Pediculus* in the middle of a small table,

around which they stood, and the parasite invariably went towards the same man, although they repeatedly changed their positions. Peacock, however, regards an instinct of preference as being very improbable. Nevertheless in my own experience among men of the same battalion living under identical conditions there were marked differences not only in the degree of lousiness, but also in the susceptibility to the toxic effects of the venom injected by the parasites.

### Morphology and Habits.

For details of the morphological structure and habits of the body-louse reference should be made to the writings of Peacock (1916), Bacot (1916), Shipley (1914) and Buxton (1939).

The female is about 4 mm. in length, the male about 3 mm. The head bears one pair of antennæ and the black eyes. The three thoracic segments are fused and present but little demarcation. There are three pairs of strong legs attached to the thorax, each of which terminates in a short powerful spine. There are eight abdominal segments, the two posterior being fused. Each of the first six bears a breathing hole, the stigma, on either side. The male is pointed posteriorly and the penis may sometimes be seen to be extruded, whereas the female's posterior is bilobed and bears a pair of ventral copulatory organs.

The parasites tend to seek shelter in the clothing, particularly that worn next the body, and prefer the seams. Warmth, humidity, shelter and freedom are, apart from ready access to the human body, the necessities for their longevity and reproduction. They feed voraciously on human blood, and adults may suck for as long as twenty minutes. The secretion from the salivary glands prevents the coagulation of the blood.

Their vitality depends on their environment. If separated from the human body, they sink into a moribund state within two days and usually die within five. Even if fed regularly and kept at body-temperature but without body-moisture, they do not survive more than a few days. Under favourable conditions their average length of life is probably about thirty to forty days after reaching maturity, the females being rather longer lived than the males.

The eggs are much more resistant and may still be capable of hatching on clothing a month after its removal from the body.

Pediculi are very prolific, and the females lay five or six eggs

per day Baoot observed one which laid a total of 295, and he estimated that a single female during her life-time may have over four thousand descendants.

The eggs are laid on the clothing and blankets, being attached to the fibres of which these are composed. They were found chiefly in the fork of the trousers and on the shirts around the armpits and on the triangles of the tail. The pleats of the kilt have already been cited as ideal shelters. It was not, however generally recognised that—under war conditions, at any rate—the *Pediculus corporis* like the *Pediculus pubis* may attach its eggs to the hair of the pubic, perineal and axillary regions. Semon and Bulhard independently drew attention to this very important fact, which I was able to confirm, as I removed some egg laden hairs from the pubic region of a man, suffering from pyogenic lesions secondary to pediculosis corporis, and incubated them on my own person. From one of the eggs a typical young body louse was hatched out.

Semon and I established the fact that in the great majority of cases with manifest *Pediculosis corporis* large numbers of ova were found on the body hair. Obviously therefore, disinfection of the clothing alone was an inadequate prophylactic measure—it was essential also to destroy the nits on the hair of the pubic and other regions.

#### The Cutaneous Lesions of Pediculosis Corporis (Figs. 44–48 Plates VII and VIII)

These may be classified as follows—

- (1) The initial urticated papules resulting from the actual bites, and the 'roseola.'
- (2) Scratch marks.
- (3) Superficial pustules.
- (4) Deep abscesses.
- (5) Circular ecthyma.
- (6) Linear impetigo or ecthyma.
- (7) Melanodermia.
- (8) Vitiligo.

In addition should be mentioned the depigmented superficial scars resulting from (5) and (6) which contrast with the surrounding melanodermia.

(1) *Urticated papules*—In a person who has recently become infested the bite of the louse provokes an urticated papule which is extremely itchy—on its subsidence an erythematous macule may

persist for a while and, if the bites have been widespread, the so-called roseola of pediculosis is seen. In one who has been lousy for a considerable time some degree of tolerance occurs, so that the local reactions at the site of the bite may not arise, and the sensation of itching diminishes. He continues, however, to scratch ("grattage instinctif")

(2) *Scratch-marks* —These, as contrasted with those of scabies, are linear, and the excoriations correspond in their different situations to the lines along which the subject can most easily scratch himself. This is well seen on the shoulders, where they tend to be oblique. On the buttocks they radiate upwards and outwards from the anus, on the outer surfaces of the thighs they run vertically upwards, on the inner surfaces upwards and outwards, and similarly on the sacrum.

(3) and (4) *Superficial pustules and deep abscesses* —In our study of pediculosis corporis Semon and I (1917) came to the conclusion that both the superficial and deep pustular lesions are formed around punctures made by the parasite. The superficial pustular lesions rapidly dry up to form crusts. The deeper lesions are abscesses of considerable depth and are often elevated and surrounded by a wide area of induration. When the small crust at the apex is removed, a minute circular orifice is revealed, through which pressure will cause the forcible expulsion of a quantity of sanguineous pus from what is evidently a bottle-shaped cavity. The pus is liquid, and there is no core, so that to label these lesions "boils" is a misnomer.

(5) and (6) *Ecthyma*. If the superficial pustules and deep abscesses are dealt with by evacuation of the pus and antiseptic applications, they usually involute, but, if left untreated, they may give rise to ecthymatous lesions. Infection evidently spreads peripherally along the lymphatics at first in circular fashion and results in the formation of encrusted ecthymatous ulcers. From these the characteristic linear ecthyma ("linear impetigo") may arise, apparently from the digital excavation by scratching of the original circular lesions. When fully formed, this type of ecthyma consists of a rectangular gutter-like ulcer covered by a thick blood-stained crust. It must not be confused with lesions superficially resembling it, which are merely a variety of "neurotic" excoriations, such as are met with in psychopathic subjects in civilian life. MacCormac and Small pointed out that excoriations of this kind



FIG. 44.—Hemorrhoids.

Observe (1) the erection of the follicles and the nasal like nodules & their pores produced by wax filling. (2) the crusted lesions resulting from secondary pyogenic infection on the lower parts of the buttocks on the lateral tuberosities.



FIG. 45.—Perianal Carcinoma.

Observe (1) the masses of follicular erection (2) the large blood-crusts covering the anal and perianal erythematous lesions, which are attacks from the excoriation of anal excoriations of infection from infected bites. (3) the radial or character of the hemorrhoids, corresponding to the direction along which the patient scratched. (4) the large erythematous ulcer on the outer side of the left buttock. (5) the hard nodule of the lower part of the tail, and anal region, both are usually spared in scabies.



FIG. 46.—Perianal Carcinoma.

In infected bites in the early stage situated in the anal or perianal area. Observe (1) the small crabs at the pores (2) the dusky (blackish-red) discoloration surrounding the lesions due to over-infection extensive of infection along the lymphatic nodular fashion.

persist for a while and, if the bites have been widespread, the so-called roseola of pediculosis is seen. In one who has been lousy for a considerable time some degree of tolerance occurs, so that the local reactions at the site of the bite may not arise, and the sensation of itching diminishes. He continues, however, to scratch ("grattage instinctif").

(2) *Scratch-marks*—These, as contrasted with those of scabies, are linear, and the excoriations correspond in their different situations to the lines along which the subject can most easily scratch himself. This is well seen on the shoulders, where they tend to be oblique. On the buttocks they radiate upwards and outwards from the anus, on the outer surfaces of the thighs they run vertically upwards, on the inner surfaces upwards and outwards, and similarly on the sacrum.

(3) and (4) *Superficial pustules and deep abscesses*—In our study of pediculosis corporis Semon and I (1917) came to the conclusion that both the superficial and deep pustular lesions are formed around punctures made by the parasite. The superficial pustular lesions rapidly dry up to form crusts. The deeper lesions are abscesses of considerable depth and are often elevated and surrounded by a wide area of induration. When the small crust at the apex is removed, a minute circular orifice is revealed, through which pressure will cause the forcible expulsion of a quantity of sanguineous pus from what is evidently a bottle-shaped cavity. The pus is liquid, and there is no core, so that to label these lesions "boils" is a misnomer.

(5) and (6) *Ecthyma*. If the superficial pustules and deep abscesses are dealt with by evacuation of the pus and antiseptic applications, they usually involute, but, if left untreated, they may give rise to ecthymatous lesions. Infection evidently spreads peripherally along the lymphatics at first in circular fashion and results in the formation of encrusted ecthymatous ulcers. From these the characteristic linear ecthyma ("linear impetigo") may arise, apparently from the digital excavation by scratching of the original circular lesions. When fully formed, this type of ecthyma consists of a rectangular gutter-like ulcer covered by a thick blood-stained crust. It must not be confused with lesions superficially resembling it, which are merely a variety of "neurotic" excoriations, such as are met with in psychopathic subjects in civilian life. MacCormac and Small pointed out that excoriations of this kind

were one of the manifestations of war neurosis, and were associated with other signs and symptoms of the psychopathic state.

A rather rare complication of the ecthymatous lesions in these cases is the development of verrucose lesions, referred to and illustrated by MacCormac (1917). The change is similar to that observed sometimes in pyogenic granuloma and in lupus verrucosus, and the lesions in question were evidently the result of the transformation of the ecthymatous ulcers into pyogenic granulomata.

(7) *Melanoderma*.—The increased pigmentation of the skin that is seen in chronic pediculosis corporis is of considerable academic interest. Histologically it is found to be due to an increase of melanin, particularly in the basal layer of the epidermis. By means of the dopa-reaction it has been shown that it is associated also with an increased number of melanoblasts.

This melanoderma is accompanied by lichenification of the skin, which becomes harsh dry, thickened and covered with excoriations surmounted by blood-stained crusts. The pigmentation is most intense on the sites of election already cited, i.e. where the patient is most inclined to scratch, but it may in severe cases become generalised varying from a *café au lait* tint to dark brown according to the situation. Contrasting with it are the linear streaks of depigmentation that occur at the sites of healed excoriations.

In rare cases of long-standing pediculosis corporis patches of pigmentation, similar to those of Addison's disease, may be seen on the buccal mucous membrane. Although the pigmentation of the skin is most intense on areas subjected to long-continued scratching, its extension to other parts including the mucous membrane of the mouth, suggests that it is in part due to the toxic action of louse-venom on the endocrine-sympathetic system and particularly on the adrenal cortex. It is possible that deficiency of vitamin C may also be a factor predisposing to the pigmentation in these cases for the dietary of those who suffer from chronic pediculosis ( *vagabond's disease* ) in civilian life is likely to be lacking in this vitamin, and our front-line troops in the last war had little opportunity of obtaining fresh fruit and vegetables. It would be interesting to observe whether the administration of ascorbic acid would have any effect on the pigmentation.

Apart, however from the pigmentation itself those in whom it is widespread and intense almost invariably suffer from profound



FIG 47—Pediculosis Corporis

Infected bites in various stages of evolution. Some are merely superficial pustules with crusting. Others are obviously deeper with larger crusts and extension of infection to the surrounding lymphatics. On the right buttock near the centre of the internatal cleft is a linear ecthymatous ulcer, and to the right of this is a linear excoriation.



FIG 49—Vitiligo and Pediculosis Corporis

Case 2—The leucoderma involved chiefly the sites of election in pediculosis corporis, viz the back, sacral region, and outer side of the thigh. In all situations it was associated with crusted ecthymatous ulcers, which on the thigh are seen to lie vertically along the direction in which the patient scratches. Linear leucodermic patches are also seen on the left thigh and the penis.

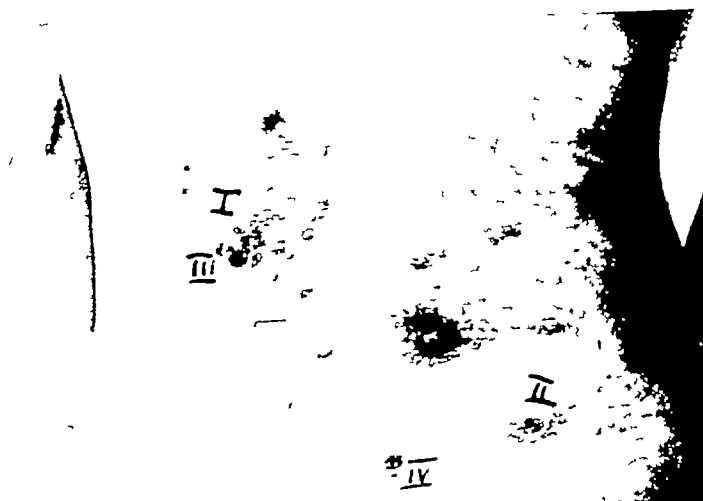


FIG 48—Pediculosis Corporis

Illustrating the localisation of infected bites to the lumbo-sacral region, and the stages in the evolution of the circular encrusted ulcer. At (I) is seen an early lesion with a superficial central pustule, at (II) the pustule has dried up to form a crust covering a small ulcer, the collarette of stripped up horny layer that frequently surrounds the infected bites in their early stages is seen, at (III) infection is spreading peripherally along the lymphatics, increasing the width and depth of the ulcer, at (IV) is a circular encrusted ulcer, from which the deeper form of linear ecthyma is produced by digital excavation and further spread of infection along the lymphatics.

source of infestation. That blankets may be a medium of transmission I can confirm from personal experience, but Peacock concludes that, although blankets, straw and paillasses may be centres of dissemination, they too are not of great importance. It is the soldier and his clothing that provide the chief source of infection. As Peacock says, "the principal conditions of transference are the long periods of proximity engendering warmth and consequent movement of the lice, as when men are compelled to sleep closely together"

**Disinfection of Men and Clothing** —For full discussions upon the prevention and destruction of lice reference should be made to the papers by Peacock (1916) Kinloch (1915) and Buxton (1939). The investigations of the first two of these observers showed that the well known N C I powder is a most effective insecticide for lice. The formula is—

Crude unwhizzed naphthalene in powder	96 parts
Creosote	2
Iodoform	2

Kinloch's experiments demonstrated (1) that crude naphthalene is more lethal than the pure form (2) that both naphthalene and creosote have a strong insecticidal action while that of iodoform is feeble, (3) that iodoform greatly increases the adhesiveness of N C I powder for cloth so that its inclusion is justified and (4) that the insecticidal power of the powder gradually diminishes on exposure to air and it should therefore be supplied in sealed tins.

Peacock advises that the powder should be dusted freely in the shirt and trousers, but should not be used in the fork as it is apt to irritate the delicate skin of the scrotum and penis. He suggests that vermijelli ointment (Soft soap 24·0 Heavy oil 70·0 Water 6·0) should be applied to the fork of the trousers, but this too may prove irritating.

As a prophylactic against lice and perhaps scabies Gunn (1917) suggested the wearing of butter muslin vests steeped in the following solution—

Naphthalene	1·0
Precipitated sulphur	1·0
Benzol	98·0

These were supplied in large numbers to officers and men on active

asthenia with low blood pressure, so that the clinical picture of Addison's disease may be closely simulated

It may be observed that the two striking changes in the skin that occur in long-standing pediculosis corporis, namely lichenification and intense pigmentation, also characterise the chronic prurigo of allergic subjects. In them, as in pediculosis, the pigmentation is deepest on the areas of prurigo, but is also generalised, and there is the same asthenia with low blood pressure and other symptoms of adrenal-sympathetic insufficiency

(8) *Vitiligo*—In the last war I collected some cases of vitiligo (leuco-melanoderma), in which the pigmentary distribution appeared to have been provoked by pediculosis corporis. As with the melanoderma, the leucodermic patches occurred first at the sites of election, e.g. on the shoulders, sacral region and outer surfaces of the thighs, but later extended to other parts, such as the penis and groins, the neck and the extremities (Fig 49).

I have elsewhere (1929) discussed the ætiology of vitiligo. The provoking factors differ in different cases, as is also the case in two closely related conditions—alopecia areata and scleroderma, but in all three a disturbance of the endocrine-sympathetic system appears to be the basic factor. They are frequently observed in association with Graves's disease, and vitiligo may precede or accompany Addison's disease.

Possibly the long-continued peripheral irritation may partly account for the development of melanoderma and vitiligo in pediculosis corporis, and in this connection it is of interest that Dore observed a group of cases of alopecia areata in which severe and long-standing irritation from pediculosis capitis preceded the loss of hair. But it is probable that in pediculosis corporis the toxic action of louse-venom on the adrenal-sympathetic system is chiefly responsible both for the generalised melanoderma and for the occasional development of vitiligo.

### **Prophylaxis and Treatment.**

From the observations of Peacock it is clear that the most important prophylactic measure against pediculosis corporis is by disinfection and other means to rid the men themselves rather than their environment of the parasites. Peacock's investigations appeared to show that the idea prevalent in the last war that certain dug-outs were swarming with lice was erroneous. A few might be found, but dug-outs could be dismissed as an important

source of infestation. That blankets may be a medium of transmission I can confirm from personal experience, but Peacock concludes that, although blankets, straw and paillasses may be centres of dissemination, they too are not of great importance. It is the soldier and his clothing that provide the chief source of infection. As Peacock says, the principal conditions of transference are the long periods of proximity engendering warmth and consequent movement of the lice, as when men are compelled to sleep closely together

**Disinfection of Men and Clothing** —For full discussions upon the prevention and destruction of lice reference should be made to the papers by Peacock (1916) Kinloch (1915) and Buxton (1939) The investigations of the first two of these observers showed that the well known N.C.I. powder is a most effective insecticide for lice The formula is—

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As a prophylactic against lice and perhaps scabies Gunn (1917) suggested the wearing of butter muslin vests steeped in the following solution—

Naphthalene	1.0
Precipitated sulphur	1.0
Benzol	98.0

These were supplied in large numbers to officers and men on active

service and proved very effective. The solution is lethal both to lice and their eggs, and clothing steeped in it provides protection for a long period.

Recently McLeod and Craufurd-Benson, working in a louse clinic in East London, have devised a powder, known as A L 63, the formula of which is for the present a secret. A garment treated with this powder will remain louse-free for about a week, and any existing lice in it are killed. At the London School of Hygiene and Tropical Medicine a liquid insecticide has been produced, a small quantity of which will render a garment louse-proof for a month (Buxton, 1941).

Disinfestation of men and clothing is carried out at divisional baths. An ordinary hot bath, with thorough lathering of the whole body is sufficient to remove the lice from the person, but it is essential also to destroy the ova on the body-hair by sponging the hairy regions with benzol or petrol. For the disinfection of clothing the Thresh disinfectors were used. With the Foden lorry "Thresh" one hundred garments per chamber are steamed at 220° F and 5 lb pressure for half an hour. Recently a new and more effective apparatus has been devised. Both lice and eggs are killed by boiling water in two minutes, and underclothing can be treated in this way. Outer clothing can be thoroughly ironed with hot irons, particularly at the seams and in the forks of the trousers where the majority of eggs are found, and then brushed with a hard brush.

The disinfected clothing should be treated with N C I powder before being donned, or, when available, the butter-muslin under-vests previously soaked in Gunn's solution should be worn.

Eggs hatch in about a week, and disinfestation should if possible be carried out at intervals of not more than a fortnight when lice are prevalent. Apart from underclothing and uniforms, blankets, great-coats and packs should be treated.

**Secondary Lesions**—The superficial and deep pustular lesions usually heal quickly if, after expressing the pus, they are painted with weak tincture of iodine. For ecthymatous ulcers Semon and I found the following ointment very effective, although it is usually held that sulphur and mercurials should not be combined—

R.	Acid Salicylic	} aa	gr 10
	Sulph Præcip		
	Ung Hydrarg Ox Flav		

Needless to say, this should not be used on sites where iodine has been previously applied

As an alternative hot compresses of perchloride lotion (1/3000) may be employed for a while, and the base of the ulcer painted daily with a 3 per cent solution of silver in spirit.

For obstinate ulcers no doubt cod liver oil either pure or in ointment form (e.g. Ung. Zinci Morrhuae B.P.C) would prove effective.

### Differential Diagnosis between Scabies and Pediculosis Corporis

It is important to recognise that the eruptions caused by the *scarus* and *pediculus* differ strikingly not only as regards the primary lesions but also in the nature and distribution of the secondary lesions. A comparison may be summarised in tabular form as follows.

#### SCABIES.

#### PEDICULOSIS

##### *Primary Lesions*

Burrows.	Urticated circular papules.
Vesicles.	Roseola.
Elongated indurated papules.	

##### *Secondary Lesions*

##### *Scratch marks*

Pin point blood-crusts at the apices of the erected follicles.	Linear excoriations.
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##### *Pyogenic lesions*

Pustules Small superficial crusts.	Superficial pustules and deep abscesses arising from the original bites. Circular and elongated encrusted ecthyma, the lesions being on the whole much larger than those of scabies.
Impetigo and sometimes ecthyma, when the secondary infection is severe. Infective eczematoid dermatitis, particularly in seborrhoeic subjects.	
Boils.	

##### *Pigmentation.*

Absent or slight in cases of long-standing	Marked, particularly at the sites of election where scratching chiefly occurs. May become intense and generalised, involving even the mucous membrane of the mouth.
	Vitiligo in cases of long-standing

*Distribution*

In some respects the distribution of the eruption caused by scabies is the reverse of that obtaining in pediculosis corporis. Thus the shoulders, back and sacral region, which are some of the sites of election in pediculosis, are the parts that are usually spared in scabies, and the hands, wrists and anterior axillary folds, which are almost always involved in scabies, are seldom affected in pediculosis.

Hands, wrists, elbows, <i>anterior</i> axillary folds, umbilicus and abdomen, lower part of the buttocks over the ischial tuber- osities	Shoulders, <i>posterior</i> axillary folds, lower part of the back and sacral region, upper parts of the buttocks, groins, thighs, and the skin between the knees and ankles
Penis and scrotum	
Front of the knees, ankles, and feet.	

It is obvious that, since in war-time infection with both parasites was not uncommon, the two clinical pictures may be superimposed

## DERMATOPHYTOSIS

In this section ringworm-infection of the groins and neighbouring parts, the axillæ, and the extremities only will be considered

*Tinea Cruris*

(*Tinea marginata*, *Eczema marginatum*, Burmese ringworm, Dhobie's itch)

This form of ringworm is caused by the *Epidermophyton inguinale* (Sabouraud), of which at least three varieties have been described. It is very prevalent in hot climates. The term "Dhobie's itch" arose from the idea that the disease was transmitted in under-clothing infected by the native washermen, who are said to stamp on the wet clothing with their bare feet before hanging it up to dry. It is very questionable, however, whether this mode of infection actually occurs. It is probable that the infection was brought to this country by persons returning from the tropics. An increased incidence was noted both after the South African War and the war of 1914-18.

### **Ætiology**

The disease is far more common in males than in females. Transmission of the infection may be direct as in the case of husband and wife, but more frequently indirect through lavatory-seats or infected clothing, such as borrowed trousers or football shorts. There is some evidence that the fungus-elements may pass through the clothing to the skin, and in one epidemic the source of infection was thought to be the chairs in the smoking room of a college.

### **Symptoms**

As a rule the earliest lesions are raised, circular papules, situated on the upper and inner surfaces of one or both thighs. These extend rapidly at their periphery and by confluence eventually form the characteristic circinate patches, the borders of which are raised and either vesicular and crusted or fringed with scales, in which the fungus may be readily demonstrated microscopically. As extension proceeds peripherally the central portions of the patches flatten and become pale or pigmented. In a typical case symmetrical, circinate plaques with festooned edges are seen spreading downwards along the inner surfaces of the thighs, upwards to the folds of the groins and over the scrotum, penis and pubic region, and backwards to the buttocks, interanal cleft and perineum. Apart however from this main bathing-drawers area, secondary patches may arise lower down on the thighs as far as the knees, in the umbilical and submammary folds, and in the axillæ. These spread in the same way and the total area involved may be very considerable. I have even seen patches on the face and pinnae. The hair of the pubis and axillæ is never invaded. A most important point is that in a considerable percentage of cases infection also extends to the interdigital spaces of the feet, which must be carefully inspected in every patient.

Moisture and heat favour the active growth of the fungus, and in hot weather particularly in obese persons who sweat freely the eruption may become very acute, extremely itchy and secondarily eczematized or infected with pyogenic cocci. Moreover as in other forms of intertrigo painful fissures may occur in the folds of the groins and interanal cleft. In cool weather the disease may become dormant the eruption consisting merely of slightly reddened, scaly patches, of which the patient may be unaware.

### Diagnosis.

Crutch-ringworm is most likely to be confused with erythrasma, intertrigo, seborrhœic dermatitis and especially psoriasis

*Erythrasma* is caused by a fungus, *Microsporon minutissimum*, which is very minute, and can be observed in potash preparations only with an oil-immersion lens. It is preferable to stain the scales after defatting them with ether. The fungus is then seen to form a dense network of fine filaments and small segments resembling spores. The eruption is usually met with in middle-aged or elderly men of all social classes, it is rare in women and is never seen in children. It seldom causes itching, and is often discovered accidentally. Its site of election is the genito-crural folds and the inner surface of the adjacent part of the thigh, and it rarely extends as far down as tinea cruris. The scrotum and pubis are hardly ever involved, but patches may occasionally be seen on the lower abdomen, under the breasts in women, and in the axillæ. It consists of light brown patches, sometimes with a rosy tint, hardly raised above the surface and only slightly scaly. Unlike tinea cruris there is no raised, festooned border and never any vesiculation. Moreover, the surface is uniform, there being no tendency for the more central parts to clear. The disease is only slightly contagious.

*Intertrigo* is the name given to an infective dermatitis, which begins in a natural fold of the skin and in uncomplicated cases is restricted to the fold and to the opposing skin-surfaces that form it. Frequently, however, the infection spreads beyond the confines of the fold, particularly in warm weather. Obesity, which increases the depth of normal folds and produces abnormal ones, is a common predisposing factor, as also is infrequent bathing. Streptococci are a common cause, and chronic cases occur in which the retroauricular, submammary, umbilical, crural and internatal folds are all involved. Intertrigo of the groins, perineum, umbilicus, and submammary folds is sometimes due to moniliasis, particularly in women with glycosuria. Seborrhœic dermatitis is a very frequent cause in the axillæ and crutch. The diagnostic points of importance are that the folds are primarily involved, and there are no raised edges at the sites of peripheral spread, whereas in tinea cruris the groin-folds are usually secondarily invaded from the upper parts of the thighs, and the eruption extends with raised margins while clearing behind them.

*Seborrhoeic dermatitis* is frequently mistaken for *tinea cruris*. Unless very inflamed and secondarily eczematized it has a fawn tint, which is also seen when it affects the axillae. As a rule patches are found on the common sites—the presternal and interscapular regions, and there is a heavy infection of the scalp with the *pitryosporon*.

*Psoriasis* frequently involves and may be confined to the natural folds in late-middle and old age particularly in obese gouty subjects of both sexes and in women at and after the climacteric. Apart from its atypical distribution the eruption in these cases is apt to become moist from serous infiltration of the epidermis and to form painful bleeding fissures in the axes of the folds. It is naturally therefore often mistaken for eczema, infective intertrigo seborrhoeic dermatitis and *tinea cruris*. The chief points of differential diagnosis are that in psoriasis the patches are more infiltrated and firmer to the touch, the edges are usually very sharply defined, but not, as in ringworm raised above the level of the rest of the patch, and, except when there is much maceration with serous oozing, the surface has a burnished appearance. The presence of patches of psoriasis elsewhere on the more usual sites will prevent an error in many cases, but they are often absent.

If there is any doubt as to the correct diagnosis, scrapings from the edges of the eruption should be examined microscopically in *Liq. potassae* for mycelial elements, which are abundant in untreated cases of *tinea cruris*.

### Treatment

Chrysarobin, or preferably dithranol (cignolin) is perhaps the most rapidly effective fungicide but it may cause an acute dermatitis of the scrotum and stain the clothing. Dithranol in *Pasta Zinci Co* (B.P.) or an ointment base 0.5 to 2 per cent may be prescribed in uncomplicated cases, providing that the patient is under close observation.

A method that I have found effective but more tedious, consists of thoroughunction at bedtime with Whitfield's ointment *Ung. Acidi Benzoici Co* (B.P.C.) and after the morning bath painting with *Liq. Iodi Mitis*, 1.0 in Spt. Meth. Ind. 2.0 and then dusting with *Pulv. Acidi Salicylici Co* (B.P.C.). This method cures the majority of cases in from ten to fourteen days and is usually well borne. In very acute cases with secondary eczematization the

patient should be completely at rest, and soothing applications, such as calamine liniment, should be employed at first

Three important points may be emphasised (1) If the infection has spread to the internatal fold, as it usually does, the patient may be unaware of it, and omit to treat it unless told to do so (2) A thorough examination should be made two or three weeks *after* the termination of treatment in order to ensure that no trace of infection remains And (3) in every case the feet, particularly the interdigital spaces, should be repeatedly inspected for signs of early infection It is advisable for the patient to apply the Pulv Acidı Salicylic Co between the toes and in the socks as a prophylactic throughout the course of treatment

### Epidermophytosis of the Extremities

("Athlete's Foot," "Toe-rot," "Hong-Kong Foot")

Since Whitfield in 1908 and Sabouraud first recognised that many scaly and vesicular eruptions in the interdigital spaces of the feet and hands and on the palms and soles are due to a ringworm infection, the condition has become a major dermatological problem The feet are nearly always infected primarily and far more commonly than the hands

#### Ætiology

In America it has been shown that the *Epidermophyton interdigitale* of Kaufmann-Wolf is the fungus responsible in about three-quarters of the cases, and the *E inguinale* (Sabouraud) and the *E rubrum* together in the remaining quarter The disease affects the more cleanly classes, being much commoner in private than in hospital practice This is doubtless because the infection is spread by the contact of bare feet with bathroom-mats and the floors of swimming-baths and changing-rooms In schools, colleges, and athletic clubs it often occurs in epidemic form As an illustration of this, Legge, Bonar and Templeton (1933) found that 51.5 per cent of new men-students and 15.3 per cent. of women-students were clinically affected Of those who attended the gymnasium for one term the percentage among males increased to 78.6 per cent, while among females the increase was only 2 per cent, the difference doubtless being partly due to the women being forbidden to walk with bare feet Templeton suggests that the greater incidence among men may also be explained by their wearing heavy shoes

and woollen socks, since sweating favours the growth of the fungus, and wool is a better medium for it than silk or cotton. Alkalinity of the sweat predisposes to its activity the optimum pH for the *E interdigitale* being 6.8 to 7. The use of alkaline soaps may favour infection of the hands.

### Symptoms

On the feet three types of the disease have been differentiated by Gray (1934) intertriginous, vesicular and hyperkeratotic. These differences depend partly on the temperature and time of year and partly on the person affected. Thus the indolent intertriginous form often becomes acute and vesicular in summer and whereas in those with hyperidrosis the eruption is likely to be moist and vesicular in ichthyotic and dry skinned subjects hyperkeratosis is the usual response to the infection.

Although on the feet some part of the sole may be the primary site of invasion in the great majority of cases the infection begins between the toes, particularly in the fourth interspace. The earliest sign is generally stripping up of the infected horny layer so that indolent scaly patches are formed, which tend to spread from the interdigital spaces outwards on the under and sometimes the upper surfaces of the feet towards the heads of the metatarsals. The edges are formed by the raised horny layer while the denuded skin within them appears reddened, glazed or actually raw. The disease may remain confined to the interdigital spaces and the parts immediately adjacent to them, or it may spread widely over the soles, producing patches, which by confluence lose their original circinate conformation and become diffuse. Frequently however and especially in hot weather and in those with hyperidrosis, the growth of the fungus becomes very active and its toxin excites a vesicular eczematoid reaction. The vesiculation tends to occur at the margins of the spreading patches, but on the soles, toes, and sides of the feet new foci may arise which are circinate and primarily vesicular. By confluence of the vesicles bullae of considerable size are formed. It is in the roofs of the vesicles that the actively growing mycelium may be seen in greatest abundance microscopically. Rupture of the vesicles and bullae is followed by the appearance of raw eczematous areas, which may be painful and incapacitate the patient. Moreover secondary infection with pyogenic cocci occurs with some frequency and acute lymphangitis of the feet and legs may result.

With the onset of cool weather the activity of the infection subsides, and the indolent, intertriginous variety of the disease remains. As already indicated, this may take the form of persistent scaliness between the toes due to constant exfoliation of the infected horny layer, or in the presence of hyperidrosis this becomes macerated, white and sodden, particularly in the fourth interspace. In dry-skinned and ichthyotic subjects there is hyperkeratosis, dry, horny and often fissured patches being formed between the toes and on the soles if these be infected.

### Complications

*Onychomycosis* —The complication most to be feared is invasion of the nails, since these remain as a source of re-infection and their treatment is difficult and unsatisfactory. It has been shown that the toe-nails may contain the fungus without themselves showing any clinical changes. The finger-nails may also become infected, and I have seen more than one instance of long-standing epidermophytosis of the feet and hands with involvement of all the finger- and toe-nails. The prognosis as regards cure is wellnigh hopeless in such cases.

*Lymphangitis* —It has already been remarked that secondary pyogenic infection may complicate the disease, especially in the acute attacks of vesiculation that occur in hot weather. Such an infection may cause acute lymphangitis of the foot and leg. More important, however, are the recurring attacks of lymphangitis that occur in some chronic cases of epidermophytosis of the feet owing to the presence of streptococcal fissures between the toes. The condition is comparable to the relapsing streptococcal lymphangitis of the face, due to fissures in the nostrils, vermilion borders of the lips, or retroauricular spaces, and, as with this, repeated attacks may lead to blockage of the lymphatics with consequent elephantiasis, which may become permanent. Very often there is little constitutional disturbance, but occasionally there may be rigors and a high temperature. The lymphangitis appears first on the dorsal surface of the foot adjacent to the interspace in which the fissure lies, and thence spreads upwards over the foot, ankle and leg. Unfortunately the origin of the condition often remains unrecognised and the primary epidermophytosis untreated, until chronic elephantiasis of the leg has become established.

*Epidermophytides* —The epidermophyton may give rise to various types of allergic reactions in the skin, as do other species of ring-

worm fungi. These reactions are termed dermatophytides and are strictly comparable to the tuberculides, syphilides and streptococoides. They depend for their occurrence upon a haematogenous dissemination of fungus-elements or their toxin from some primary focus of infection, and an allergic hypersensitivity of the skin. The eruptions are termed trichophytides, microsporides, favides and epidermophytides according to the kind of fungus responsible. They vary in type like the tuberculides, syphilides and streptococoides. The following have been described a lichenoid follicular eruption comparable to lichen scrofulosorum, the follicular syphilide, and the lichenoid streptococoides an eczematoid eruption comparable to the eczematoid tuberculide and streptococoides erythema multiforme erythema nodosum erythema scarlatiniforme (Barber 1929). Just as patients with tuberculide eruptions gave positive cutaneous reactions to intra dermal injections of tuberculin, so those who have or have had ringworm infection will react similarly to injections of certain fungus-extracts, which may be conveniently termed trichophytin. Both with tuberculin and trichophytin eczematoid patches may develop after a few days at the test sites, particularly in patients with the eczematoid tuberculide and the eczematoid dermatophytide.

In epidermophytosis the commonest dermatophytide is the eczematous one. Frequently it takes the form of a vesicular eruption on the hands, particularly along the sides of the fingers, which by many observers is considered indistinguishable from dyshidrosis or cheiropompholyx. Some believe that in nearly all cases of the condition formerly designated by these terms the eruption is really a vesicular dermatophytide or actually caused by the presence of fungus elements. The great majority of observers agree that even in cases that might be regarded as true dyshidrosis the vesicles bear no relationship to the sweat-ducts, being in fact comparable in their formation to those of eczema. My personal opinion is that the confusion and the contradictory opinions that have existed on this subject since Tilbury Fox first described what he termed dyshidrosis and Hutchinson later labelled cheiropompholyx, are entirely due to faulty clinical observations and descriptions. I agree that most vesicular eruptions of the hands and feet are independent of the sweat glands, and are eczematoid in type both clinically and histologically. Their causation is often difficult to determine with certainty. They may be due to external

irritants, to infection with and sensitisation of the skin to pyogenic organisms (infective eczematoid dermatitis), to the absorption of the toxins of such organisms either in the skin itself or in some other focus, or an allergic reaction to other circulating antigens, e g drugs, or part of the recurring eczema that obtains in persons with multiple allergic symptoms, and, as has been said, they are frequently a dermatophytide

Nevertheless there is a true dysidrosis, which occurs only in those with hyperidrosis of the hands and feet, and which affects the palms and soles rather than the lateral surfaces of the digits. The vesicles at first are minute, closely set in patches, and have the true translucent, sago-grain appearance. This eruption has an obvious association with the sweat-ducts.

Another form of eczematoid epidermophytide, which is not very uncommon, is that which occurs chiefly around the ankles and on the legs in the form of circular patches similar to nummular eczema due to other causes. In patients with this condition an intra-dermal injection of trichophytin will be followed in a few days by the appearance at the injection site of a circular eczematoid patch exactly comparable to those composing the original eruption. When the infection of the feet is successfully treated, the eczematous patches disappear spontaneously. Goldsmith (1931) has described a case of erythema multiforme, which was almost certainly a dermatophytide due to a laboratory-infection with the *Epidermophyton interdigitale*.

### Diagnosis

In the great majority of cases of intertriginous dermatitis between the toes one of the varieties of the above-mentioned ringworm fungi is responsible. A similar condition, however, in the interdigital spaces of both the hands and feet is also caused by *monilia infections* ("erosio interdigitalis blastomycetica"). The lesions often consist of white sodden-looking skin with central fissures. Extension to the backs of the hands or over the feet may occur, reddened patches with scaly margins and vesico-pustules being found. On the fingers this monilial infection occurs chiefly in those who frequently wet their hands, since moisture favours the growth of the fungus, and on the feet in those with hyperidrosis. Dowling (1930) and others have reproduced similar eruptions by planting cultures of *Monilia albicans* between the digits and elsewhere. Hyperidrosis *per se* may produce maceration of the horny

layer between the toes so that it becomes whitish and sodden a similar appearance may also be seen in severe cases on the soles, particularly on the heels. Eczema of non-infective origin and psoriasis may of course affect the interdigital spaces, and, as in *tinea cruris*, the latter is particularly likely to cause an error of diagnosis.

The eruptions, which in the past have often been wrongly attributed to fungus-infection, even by dermatologists, are *pustular bacteride* and *pustular psoriasis of the extremities*. These conditions are aetiologicaly identical in that they are due to a focal infection with a streptococcus, the chief focus as a rule, being in the tonsils. The difference between the two depends upon whether or not the patient is a psoriatic. Clinton Andrews (1934) has described *pustular bacterides* as follows. They are chronic recalcitrant vesicular and pustular eruptions, symmetrically located on the palms and soles, which during quiescent periods become dry erythematous, exfoliative patches. The course is characterised by repeated exacerbations, during which groups of vesicles or pustules break out over the entire involved areas with severe itching and sometimes swelling and pain. From day to day fresh groups of lesions may appear and in some cases vesiculation is pronounced, whereas in others the lesions are almost entirely pustular from the onset. In still others the early vesicles become pustules in the course of time. Gradually the number of new lesions diminishes and the condition temporarily subsides to a quiescent stage in which the involved areas are diffusely erythematous, dry shiny and exfoliative."

In *pustular psoriasis of the extremities* the eruption may occur alone without lesions of psoriasis elsewhere or it may be associated with such lesions, some of which may also develop occasional pustules. The patches are dry red, scaly and usually sharply defined as in ordinary psoriasis, and in them pustules of varying size, which dry up to form characteristic brown scabs, are formed. In both *pustular bacteride* and *pustular psoriasis* the sites of election are the thenar and hypothenar eminences and the central parts of the soles. Although these eruptions tend to be symmetrical, they may occur unilaterally or asymmetrically and single patches are met with. Both eruptions may involve the fingers and toes. *Pustular bacteride* is more vesicular in type and histologically resembles the vesicular epidermophytides, whereas *pustular*

irritants, to infection with and sensitisation of the skin to pyogenic organisms (infective eczematoid dermatitis), to the absorption of the toxins of such organisms either in the skin itself or in some other focus, or an allergic reaction to other circulating antigens, e g drugs, or part of the recurring eczema that obtains in persons with multiple allergic symptoms, and, as has been said, they are frequently a dermatophytide

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I regard a preliminary treatment designed to exfoliate the thickened horny layer of the feet, particularly between and around the toes, as essential in chronic cases, and in the early stages such treatment alone may suffice to cure. By this method a great deal of the fungus must obviously be removed in the exfoliated horny layer and any that remains lies on the surface exposed to attack by whatever fungicide is chosen. If well tolerated, this preliminary treatment is continued until the denuded skin resembles that of a baby's foot and for this a period of two to three weeks is usually required. A lotion of *Acidi Salicylici* dm. 1, Spt Meth. Indust to oz. 1, is applied freely and vigorously between and around all the toes of both feet and over the soles morning and evening. After each application the *Pulv Acidi Salicylici Co* (B.P.C.) should be sprinkled between the toes, over the feet and in the morning into the socks or stockings. Various fungicides may then be employed to complete the cure. Dithranol (cignolin) as in *tinea cruris*, is very effective, but must be used with caution, since it may cause an acute dermatitis. It may be prescribed in an ointment or paste, or as a paint dissolved in benzol (0.5-2 per cent.) A safer application is carbol fuchsin paint (saturated solution of fuchsin in absolute alcohol, 10 c.cm. with 5 per cent carbolic lotion, 100 c.cm. mixed and filtered. After two hours add boric acid 5 grms. After the next two hours add resorcinol 10 grms. in acetone, 5 c.cm.) This should be painted on freely once or twice daily and the *Pulv Acidi Salicylici Co.* applied between the toes, over the feet and into the socks or stockings every morning. The carbol fuchsin paint occasionally causes a dermatitis but if well borne it may be continued for a month. The powder should then be used alone for some weeks and the feet inspected again. If the patient is apparently cured, he should be advised to continue the morning application of the powder indefinitely. In view of its acidity it will tend to prevent relapse from any fungus-elements that may have escaped destruction or re-infection, since the fungus prefers an alkaline medium for its active growth. After the preliminary exfoliating treatment I frequently employ the method outlined for the treatment of *tinea cruris*—the application of Whitfield's ointment at bedtime and of iodine and the powder in the morning. The following are the instructions issued to patients. The ointment should be thoroughly rubbed into the feet particularly between the toes, at night time. This

psoriasis is drier, almost always purely pustular and not vesicular, and the histology is that of psoriasis with superadded pustule formation. During the exacerbations of pustulation there is usually a considerable leucocytosis. Cultures from the pustules are invariably sterile apart from occasional colonies of *Staph albus*.

On the hands active infection with the epidermophyton, as opposed to the commoner epidermophytides, presents different clinical appearances according to the activity of the fungus and the individual affected, and a certain diagnosis may be impossible without a microscopical examination of scrapings. As on the feet, the interdigital spaces may be first involved, and thence the infection spreads along the sides of the fingers and on to the dorsal and palmar surfaces of the hand. The eruption may be dry and consist of reddened patches bordered by a fringe of the stripped-up horny layer, or, particularly in hot weather, it may be frankly eczematous with vesiculation, serous oozing and crusting. In either case the circinate configuration of the patches will suggest a fungus infection. In patients, however, in whom the disease is of long standing, the eruption may become diffuse, particularly on the palms, which merely appear red and scaly. In dry-skinned persons and ichthyotics the infection is likely to provoke hyperkeratosis with splitting of the thickened horny layer and the formation of painful fissures. In those with hyperidrosis the horny layer may be sodden and macerated, particularly in the interdigital spaces and on the sides of the fingers.

### Treatment

Most dermatologists rightly consider that epidermophytosis of the feet, if of long standing, is extremely difficult to cure, and if the nails are extensively involved, it may be almost impossible unless the nail-matrices are destroyed. The chief reason for the persistence of the infection on the feet, apart from involvement of the nails, is the thickness of the horny layer. Other factors that favour the growth of the fungus in this situation are the wearing of woollen socks and heavy shoes, particularly if these have rubber soles, hyperidrosis, and the compression of the interdigital spaces by shoe-pressure, since, like other fungi, the epidermophyton thrives in moisture, warmth, and unaërated folds. Successful treatment of the hands presents much less difficulty. It is quite likely, however, that many apparent relapses in foot-ringworm are really re-infections.

### Prophylaxis

Patients who have had epidermophytosis of the feet should be advised to continue to use the Pulv. Acidi Saheylici Co. as a foot-powder after apparent cure. There is an obvious risk of re-infection from bedroom slippers, which if made of felt are best discarded. Leather shoes should be disinfected with formalin or by swabbing with a lysol solution (1 in 10) several times during the course of the treatment. Patients should be warned of the risk of infection from bath mats, changing rooms and swimming baths, and enquiries should always be made as to whether other members of the household may be infected. In schools, colleges, athletic-clubs, ablution centres, swimming baths and bathing pools foot-baths of a 2 per cent. solution of sodium hypochlorite should be provided and members should be enjoined to soak their feet in these before dressing. This prophylactic measure has been made compulsory in such institutions in America with very good effect.

### IMPETIGO CONTAGIOSA

In the last war seborrhoeic dermatitis, complicated by a secondary invasion by streptococci and staphylococci of the *aureus* type, was extremely common. Like pediculosis corporis and scabies it was responsible for a serious wastage of man power and was difficult to treat successfully under existing conditions, and was very prone to relapse. It was often diagnosed as impetigo but differed aetiological and clinically from impetigo contagiosa. Among the Expeditionary Force in France during the earlier part of this war the seborrhoeic state was met with not more frequently than in civilian life, whereas impetigo contagiosa of the usual type affecting the face and scalp was very common.

In the great majority of cases impetigo is due to a superficial infection of the skin with *Strept. pyogenes longus* but a rapid secondary invasion of the lesions with *Staph. aureus* always occurs. In one form of bullous impetigo however and in pemphigus neonatorum the latter organism is primarily responsible. Here the streptococcal variety will alone be described.

Infection with the streptococcus takes place just beneath the horny layer. The primary lesion is a minute red spot, which very rapidly becomes a superficial vesicle or bulla owing to the exudation

should be done with a piece of lint and not with the fingers. Cotton socks should be worn during the night. The iodine should be carefully painted over the feet, particularly between the toes, after washing off the ointment in the morning, and the powder freely sprinkled over the feet and between the toes after using the iodine, and also into the socks or stockings worn during the day. The above suggestions for treatment are suitable in the great majority of patients, but in very acute cases with eczematisation or severe secondary pyogenic infection weak antiseptic footbaths (potassium permanganate 1/4,000) and soothing applications, such as calamine liniment, should first be prescribed.

A method of treatment, to which considerable attention has been paid recently even in the lay-press, is by applying a mixture of pure phenol and camphor (Francis, 1941). I have no personal experience of it. It would appear to be dangerous, unless the patient is treated in bed and every precaution is taken to ensure that the treated surface remains dry, since in the presence of water or excess of sweat the phenol is released and may cause tissue-necrosis.

For the hands the same exfoliating lotion may be first used on the palms, if there is marked hyperkeratosis, until the thickened horny layer is removed, but thorough inunction with Whitfield's ointment at night time with the patient sleeping in gloves is usually sufficient.

The treatment of ringworm of the nails is most unsatisfactory. The method that offers the best chance of a cure is to remove the affected nails under a general or local anæsthetic, and to scrape the exposed nail-beds, which should then be dressed daily with a fungicide until the new nails have grown. A solution of dithranol—2 per cent in benzol—and the carbol-fuchsin paint are probably the best applications. Unfortunately the new nails become infected in a high percentage of cases. There is some evidence that treatment of the nail-beds after removal of the nails with fractional doses of x-rays increases the percentage of cures, and I have seen a case in which such treatment alone without removal of the nails was curative. Jacobson and Corsi (1943) have succeeded in curing a few cases by painting the nails weekly with thorium-X in spirit, (1,000 to 2,000 units to the cm) over a period of some months, but in other cases the treatment was without effect.

fissures at the angles of the nostrils or mouth. Their occurrence is predisposed to by the use of strongly alkaline soaps, which damage the protective horny layer but such soaps may of themselves give rise to dry, scaly patches apart from infection. Impetigo pityrodes is contagious, and many epidemics of it have been described. It has to be differentiated from the scaly patches caused by the pityrosporon of *Malassez*, which are usually seen in older children and adults, and from the eczematoid tubercle.

### Ætiology

The primary phlyctenule of streptococcal impetigo rapidly becomes secondarily invaded by the staphylococci always present in the mouths of the pilo-sebaceous follicles and as these organisms grow much more readily and abundantly than streptococci in ordinary media, cultures made even from early lesions of impetigo may give pure growths of staphylococci. In order to obtain the streptococcus the serum from the early phlyctenule or from the raw surface exposed after removal of a crust is drawn up into a small glass pipette half full of serum broth the ends are sealed and the pipette incubated at 39° for about eight hours. If the lower end of the pipette be then broken, the first few drops of the medium will be found to contain the streptococcus in pure culture, whereas the fluid in the upper part in contact with air will contain staphylococci. The streptococcus, being a facultative anaerobe will grow alone in the lowest portion of the pipette. When the phlyctenules or bullæ of impetigo occur on the palmar surface of the hand, where there are no pilo-sebaceous follicles and consequently staphylococci are less numerous, secondary staphylococcal invasion occurs relatively late the serous contents of the phlyctenule tend to remain clear and the streptococcus may be obtained in pure culture even on ordinary media. Culture media containing crystal violet which inhibits the growth of staphylococci, may also be used. By this means abundant growths of *Strept. longus* in ordinary impetigo and ecthyma are obtained, whereas in cultures from normal skin and from various other skin eruptions not of streptococcal origin either no streptococci grow at all or only a few isolated colonies. Sabouraud's contention as to the streptococcal causation of impetigo pityrodes is also thus confirmed.

The chief factors predisposing to impetigo are trauma of all kinds whereby the horny layer of the skin is damaged, and exposure to infection with virulent streptococci. As its name implies, it is

of fluid, and these may be considered as the characteristic lesions of the disease. Where the horny layer is thin, as on the face, they are flaccid, but in other parts, particularly on the palms and soles, they may be tense. In the early stage the contained fluid is clear serum, and from it the streptococcus may often be grown in pure culture, but secondary infection with staphylococci soon takes place, and the fluid then becomes cloudy from the presence of leucocytes; in this stage the vesicles are surrounded by a red halo of hyperæmia. The fluid may escape owing to rupture of the roof of the vesicle, or it may dry up spontaneously. In any case the dried serum forms characteristic crusts, which are yellow or amber-coloured, or sometimes brown from admixture with blood or dirt. After a few days the crusts fall, leaving a pink stain to mark the site of the original lesion. Should a crust be removed before healing has taken place beneath it, a raw, oozing, and sometimes bleeding surface is exposed, which soon becomes encrusted again. Healing takes place by complete regrowth of normal epithelium, and no scar is left. The subjective symptoms are usually slight, but there may be some itching or burning, and in children fresh lesions are produced by scratching. Various types of impetigo contagiosa have been described.

In *impetigo circinata* there is comparatively little exudation, and the lesions tend to spread peripherally while healing in the centre, thus forming circinate patches with crusted margins, the condition is often mistaken for circinate ringworm. By confluence of the patches extensive gyrate figures may result (*I. gyrata*).

In *impetigo bullosa* the lesions form large bullæ, the roofs of which are flaccid, except where the horny layer is thick, as on the palms and soles.

*Impetigo ptyrodes* is, as Sabouraud insisted in 1904, a dry, attenuated form of streptococcal impetigo, situated as a rule on the face, in which the lesions consist of dry, furfuraceous patches usually of irregular shape. They correspond probably to the majority of cases of *dartre volante* or tetter of older authors. They are met with towards the end of an attack of ordinary impetigo, and one may see in the same patient all stages from the crusted, oozing lesions to the dry, scaly patches referred to. They are, however, also very common in children suffering from chronic nasal catarrh, in whom there has been no definite attack of ordinary impetigo, and are then frequently associated with streptococcal

are immiscible with serum. In this stage the object is to cause desiccation of the lesions. A method that is as a rule very effective consists of swabbing all the affected areas several times during the day with a solution of perchloride or oxycyanide of mercury (1 in 3 000) in 50-75 per cent. spirit, and at bedtime applying

R $\bar{y}$ Resorcin	gr 10
Calaminæ Præpt.	gr 20
Zinci Oxidi	gr 20
Sodii Biborat.	gr 10
Glycerini	m. 15
Spt. Vini Meth. Indust.	m. 30
Aq Calceæ ad	$\bar{3}$ l

Under this treatment the eruption dries up, the moist crusts drop off, and scaly patches remain. The lotions may then be discontinued and a mercurial paste substituted, e.g

R $\bar{y}$ Hydrarg. Ammon. vel Hydrarg. Oxidi Flav	gr 5-10
Past. Zinci Comp (B.P) ad	$\bar{3}$ l

An alternative method is to apply Eau d'Alibour instead of the mercurial lotion, e.g

R $\bar{y}$ Zinci Sulphatis	gr 6
Cupri Sulphatis	gr 4
Aq Camphoræ ad	$\bar{3}$ l

The dyes, such as crystal violet, acriflavine, and brilliant green, are very effective but are unsuitable for the treatment of ambulatory cases of facial impetigo.

The emulsifying bases have not the disadvantages of the usual lanoline and paraffin bases. They have a drying effect on the skin and afford a suitable vehicle for antiseptics in the early moist stage of impetigo. Mumford (1940) recommends the following modification of Ung. Quinolor (Squibb) both for impetigo and sycosis

1 Thymol	0.1
2 Benzoyl peroxide	10.0
3 Industrial spirit	10.0
4 Chmosol	0.5
5 H.E.B. simplex	25.0
6 Water to	100.0

(Rub number 1 in 3 add 2, and then add 4 and 5 and finally water) He also suggests Acriflavine 1/1000 in water with 12½ per cent. H.E.B. Simplex and thymol 1/1000 (warm and emulsify) for

contagious and is apt to be communicated from one member of a family to another by indirect and direct contact. It also occurs in epidemic form in schools and other communities. The so-called *scrum-pox* is impetigo contagiosa spread by contact on the football field. Apart from contagion it may arise spontaneously and is apt to complicate itching conditions, such as scabies and particularly pediculosis capitis. Impetigo of the scalp is frequently due to the latter cause. It may begin around the nostrils or on the upper lip by infection of the skin with a streptococcus derived from an acute rhinitis or purulent nasal discharge, and around the ear from the discharge of a chronic otitis media. It frequently arises from fissures at the angle of the mouth (*la perlèche*).

### Morbid Anatomy.

Microscopical section through a vesicle shows that this is situated at the level of the stratum granulosum, so that the roof is formed by the stratum corneum and the base by the upper part of the prickle-cell layer. The fluid consists of serum containing leucocytes, chains of streptococci may be seen along the floor of the vesicle and groups of staphylococci near the roof and sides. The epidermis beneath is œdematous, and leucocytes may be seen in the intra-epithelial lymph channels. The capillaries in the papillæ are dilated and there is slight inflammatory infiltration.

### Prognosis.

The prognosis varies according to the individual case. An acute attack of the disease occurring in a child or adult otherwise in good health tends to run a definite course of some three to six weeks' duration, recovery taking place spontaneously owing to the gradual development of immunity to the infecting streptococcus. On the other hand, in debilitated persons, particularly in those who suffer from a chronic streptococcal infection of their mucous membranes, so common among children of the poorer classes, or from otorrhœa, impetigo, usually associated with streptococcal fissures and intertrigo, may become a chronic disease. It is in these cases that, apart from fissures at the muco-cutaneous junctions and in the natural folds of the skin, the dry scaly form of impetigo already described is likely to occur.

### Treatment.

*Local* —It cannot be too strongly emphasised that the application of antiseptics incorporated in the usual ointment bases is fundamentally wrong during the active stage of the disease, since such bases

at the same times for three days. The patient is instructed to drink at least five pints of bland fluid per diem during the course.

It is advisable to supplement the diet with preparations rich in vitamin A and with vitamin C since there is some evidence that these increase the resistance of the skin to pyogenic infection. Moreover, as some of the toxic effects of the sulphanilamide group of drugs depend upon damage to the liver cells I always prescribe these drugs with considerable amounts of vitamin A. In the flushed or plethoric type of seborrhoeic patient in whom as already said, acute eczematization of the affected areas is likely to occur full doses of alkalis internally and calamine liniment locally should be prescribed until the eruption has dried up a weak mercurial paste or the acriflavine emulsion being then substituted for the liniment.

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impetigo Sulphathiozole, 5 per cent in Pasta Zinci Co B.P or an oil-in-water emulsion affords a clean and often rapidly effective treatment It is important to remove crusts before each application.

These methods are suitable for uncomplicated impetigo, but in some cases a secondary eczematization of the affected areas occurs owing to sensitisation of the skin to the pyogenic cocci Such cases are as a rule either seborrhœic or allergic subjects and the usual antiseptics are not tolerated For them the calamine liniment, recommended for impetiginised seborrhœic dermatitis, is effective and should be combined with suitable internal treatment

When the eruption is widespread, baths containing zinc sulphate are indicated, a quarter of a pound of the salt being added to thirty gallons of water In resistant cases of impetigo local applications to the infected areas of ultra-violet light rays, combined with total light baths, are of value.

Before a patient is passed as cured he should be carefully examined for streptococcal fissures at the muco-cutaneous junctions and in the natural folds, and, if present, these should be treated by painting them twice daily with a 3 per cent solution of silver nitrate in spirit and applying a mercurial paste

*Internal and general treatment*—It is sometimes taught that no treatment other than local measures is necessary in impetigo, but in some cases this is certainly untrue The disease often runs the course of an acute infection, and in the majority of cases recovery would doubtless occur without any treatment whatever owing to the development of immunity to the infecting organisms Cases are met with in which despite assiduous and skilful local treatment new lesions continue to appear In them change of environment, particularly to a bracing place by the sea, may lead to rapid recovery. In the last war I had a very severe attack in Mesopotamia during the hottest time of the year, and no method of treatment that I employed had any permanent effect, but on my return to the relatively cool climate of Bombay the eruption quickly subsided

In every case the patient should be examined for chronic foci of infection elsewhere than in the skin, since their presence may retard recovery or favour relapse

*Sulphonamide therapy* In severe or resistant cases of impetigo it is justifiable to employ one of the sulphonamides internally, but only for a few days I prefer sulphathiozole, and prescribe 2 grm after breakfast and tea and at bedtime for two days, and 1.5 grm

in urine, the composition of which fortunately rendered it a useful antidote. In the April attack there were 15 000 casualties, 5 000 of which were fatal, and many prisoners were taken by the Germans. Another gas attack was made early in May the men had by then been provided with respirators, consisting of cotton pads soaked in a solution of sodium thiosulphate, sodium bicarbonate and glycerine, and these gave some protection. The third attack on May 24th and all subsequent ones produced less serious results, as respirators of steadily increasing efficiency were introduced. From December 1915 phosgene was used as well as chlorine but after August 1916 there were no further cloud gas attacks. Gas shells were introduced in the autumn of 1916 they contained phosgene, chloro-pyridin, xylyl and benzyl bromide, and allied lung irritants. In July 1917 'yellow cross' shells containing mustard gas and still later organic arsenic compounds were used by the Germans. Different types of shells were generally employed simultaneously. Those containing arsenical compounds were expected to produce such irritation that the men would discard their respirators and so make it possible for phosgene from other shells to act. Hand grenades containing chloroacetone, chlor-sulphonic acid and other poisonous gases were also used. Mustard gas caused eight times as many casualties among the allied troops as all the other poison gases together but the number of fatal cases never exceeded 2 per cent.

In spite of much research in chemical warfare it is remarkable that even now the two most effective gases belong to the phosgene and mustard gas groups used in the last war. The respirators provided, both for soldiers and for civilians give complete protection for the eyes, nose, face and lungs against every type of gas likely to be used.

## LUNG IRRITANT (ASPHYXIAN) GASES

### CHLORINE AND PHOSGENE

The first effect produced by the irritant action of chlorine and phosgene,  $\text{COCl}_2$ , is a profuse exudation of a thin, pale yellow albuminous fluid by the bronchial mucous membrane, as well as a very active secretion by the lachrymal and salivary glands these are the results of protective reflexes, the object of which is to dilute the irritant poison and render it innocuous. At the same time spasm

## CHAPTER XXIX

### GAS POISONING

Thucydides describes how in the Peloponnesian War in the fifth century B.C. Archidamos, son of the King of the Lacedemonians, ordered faggots impregnated with pitch and sulphur to be set on fire outside the walls of a besieged city. The smoke carried by the wind rendered one side of the defences untenable. A thunderstorm then broke and the rain extinguished the fire and so led to the failure of the first recorded gas attack. In 1499, when the Turks had passed the Isonzo and were threatening Venice, Leonardo da Vinci advised the Venetians to throw poisonous powders, such as yellow arsenic and verdigris, from the topmasts of their ships so as to choke the enemy. Fifty years later red Indians in Canada fortified their cabins with faggots of wood and feathers smeared with the grease of the sea wolf and set fire to them on the approach of the French, many of whom were killed by the thick, black, stinking smoke. In 1812 Lord Dundonald submitted to the Prince Regent secret plans for the use of asphyxiating gas. Though regarded "infallible and invisible," its use was not sanctioned, and the plan was again rejected when suggested for use against the French fleet when war seemed imminent in 1840, because it "would not accord with the principles of civilised warfare," and because it was "too terrible for use by a civilised community."

The use of poison gas was expressly forbidden by the Hague Convention of 1899. The Germans, who had signed the Convention, attempted to excuse their violation of it in 1915 by stating through their official news in July 1918 that the idea was originated by the British Admiral, Lord Dundonald, but they failed to add that it was repeatedly rejected by the British Government on the score of inhumanity.

Chlorine was first used on April 22nd, 1915, in a German attack on Algerian and Zouave troops. The gas was set free from cylinders in the enemy trenches to drift with the wind. On the two following days attacks under cover of gas were made on Canadian and English soldiers in the neighbourhood of Ypres. Although no respirators were available and the losses were heavy, the troops held their ground. A few men improvised respirators by soaking handkerchiefs

due to the asphyxia and partly to the irritant action of chlorine dissolved in swallowed saliva and nasal and bronchial secretion.

### Symptoms

Lewis Freeman, a Canadian soldier who was taken prisoner after being wounded and gassed in the attack near Ypres on April 23rd 1915 and subsequently escaped from Germany, gave the following graphic description of his symptoms in the *Cornhill Magazine* for February 1917

"I shall never forget the horrible agony of surprise in the eyes of the men who got that first dose. It was the look of a dog being suddenly beaten for something it hadn't done. They looked at each other with questioning eyes—I only recall hearing one man start cursing—then they began gulping and coughing and then fell down with their faces in their hands. My first sensation was of a smarting away up made of my nose this quickly extended to my throat, and then as my lungs suddenly seemed filled with red hot needles I was seized with a spasm of coughing. Coughing up red hot needles is not exactly a pleasant operation, and the pain was intense. Mercifully it was only a few minutes before a sort of stupor seemed to come on, but even as I passed into half unconsciousness I was aware of my outraged lungs revolting in heaven that shook my frame against the poison that had swamped the trench. With some of my comrades the fighting instinct was the last thing that died, and I have a sort of recollection of two or three of them clutching at the parapet and firing from cough-shaken shoulders off into the depths of the rolling yellow gas clouds. I had rolled and writhed, in the agony of the pain of the gas in my lungs, in a pool of slush in the bottom of the trench, and it must have been the lying with my face buried in the shoulder of my wet woollen tunic that saved my life. Most of my comrades were quite unconscious when the Huns, with their heads protected by baggy anods, came pouring into the trench, but I had enough of my senses left unparalysed to be able to watch them in a hazy sort of way

The sensation of suffocation, which follows the initial burning pain in the nose, throat and eyes, is accompanied by pain, which is often severe, in the chest, especially behind the sternum. The irritation of the throat leads to coughing and that of the eyes to profuse lachrymation. Respiration becomes painful rapid and difficult. Retching is common and may be followed by vomiting, which gives temporary relief. The lips and mouth are parched and the tongue is covered with a thick dry fur. Severe headache rapidly follows with a feeling of great weakness in the legs. If the patient gives way to this and lies down, he is likely to inhale still more chlorine, as the heavy gas is most concentrated near the

of the bronchial muscles occurs in an attempt to obstruct the passage of the gas into the alveoli. In severe cases the bronchial secretion and spasm not only fail to protect the alveoli, but obstruct the entry of air into the lungs to such an extent that the patient becomes asphyxiated and may die before the fluid is expectorated and the spasm relaxes. An autopsy at this stage shows slight congestion of the larynx and severe congestion and œdema of the trachea and larger bronchi, which are filled with frothy fluid. Gas reaching the alveoli damages the capillaries which become dilated and engorged. Thrombosis occurs in some, whilst exudation of plasma into the air-space occurs from those through which blood continues to flow with production of pulmonary œdema. The lungs are intensely congested and œdematous, but the violent respiration caused by the asphyxia produces small patches of over-distended lung, seen on the surface as light-grey areas in the least damaged parts, into which air can still pass. The distended alveoli may rupture into the interstitial tissue, and air may spread into the mediastinum and even to the neck.

In all but the mildest cases the asphyxial stage is followed by a stage in which acute inflammation with profuse exudation of lymph occurs as a result of the irritant action of the gas on the bronchial mucous membrane and the alveoli. If the patient dies in this stage the serous fluid in the bronchi is replaced by muco-pus, and more or less extensive broncho-pneumonia is found.

There is no conclusive evidence that the chlorine is absorbed by the blood and conveyed by it to other parts of the body. Nephritis is occasionally found *post mortem*, though there is hardly ever any clinical evidence of its presence, thus albumin and casts are rarely found, œdema never occurs, and only one case of uræmia was recorded. According to Leonard Hill, the "nephritis" was not due to the toxic action of the gas after absorption, but was a result of asphyxiation and analogous to the condition which results from temporary occlusion of the renal artery. At a later stage secondary toxic effects may be caused by absorption of the products of the pathological changes in the lungs. If death occurs in the earlier stages, the right side of the heart is greatly dilated and the brain and all the abdominal organs show much congestion due to asphyxia. The mucous membrane of the stomach is red and covered with thick yellowish mucus, submucous hæmorrhages are common, and superficial erosions may be present. These changes are partly

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ground. In severe poisoning unconsciousness follows; the face assumes a pale greenish-yellow colour and death occurs shortly afterwards. When a man lives long enough to be admitted into a clearing station, he is conscious but restless, his face is violet red and his ears and finger nails blue, his expression is strained and anxious as he gasps for breath. He tries to get relief by sitting up with his head thrown back, or he lies in an exhausted condition, sometimes on his side with his head over the edge of the stretcher in order to help the escape of fluid from the lungs. His skin is cold and his temperature subnormal, the pulse is full and rarely over 100. Respiration is jerky, shallow and rapid, the rate being often over 40 and sometimes even 80 a minute, all the auxiliary muscles come into play, the chest being over-distended at the height of inspiration and, as in asthma, only slightly less distended in extreme expiration. Frequent and painful coughing occurs and some frothy sputum is brought up. The lungs are less resonant than normal, but not actually dull, and fine râles with occasional rhonchi and harsh but not bronchial breathing are heard, especially over the back and sides.

Headache is generally severe, and there is also considerable epigastric discomfort, due partly to the strain of coughing and partly to gastric irritation, as it is increased if an attempt is made to eat.

The intense dyspnoea of this asphyxial stage lasts about thirty-six hours, after which it gradually subsides, if death does not occur before. The patient, exhausted from his fight for breath, then falls asleep and wakes up feeling much relieved.

A few hours later acute bronchitis or broncho-pneumonia develops. In severe cases the quiescent interval is short and the bronchitis very severe. The sputum is now viscid, yellow or greenish and muco-purulent with occasional streaks of blood. Respiration becomes more shallow and rapid, and the rate may finally be even 70 or 80 a minute. The pulse is small and very rapid, the temperature rises and is often as high as  $104^{\circ}$ . The patient may now become delirious. Pleurisy may occur, and in some instances empyema and gangrene of the lung follow.

After recovery from the bronchitis and pneumonia the patient remains weak and exhausted for a considerable time. He gets tired very rapidly and is unable to walk quickly or uphill without getting short of breath, even after the last signs of bronchitis have disappeared. Hunt and Price-Jones (1919) showed that most

late cases could be grouped into two distinct classes, the remainder being mixed and showing symptoms belonging to both. In the more common class the symptoms are almost indistinguishable from those described as 'the effort syndrome' the patients complain of precordial pain and become rapidly exhausted and dyspnoeic with persistent tachycardia after comparatively little exercise. The other class is characterised by the occurrence of nocturnal attacks of dyspnoea, which last from five to thirty minutes and may be repeated several times in one night. The patient sits up and breathes rapidly but with much less difficulty than in asthma, and there is little or no cyanosis there is no constant change in the pulse and no abnormal physical signs are present in the chest. Exercise does not give rise to dyspnoea nor any abnormal increase in the pulse rate. In both groups the patients complain of head ache, which is often accompanied by vertigo. Epigastric pain which is rarely severe and is worse after meals, is common, and pain may be felt across the chest especially over the heart in most cases only after exercise. Pyrexia without any obvious cause may occur. Bronchitis is occasionally present, but there is no emphysema. The heart may be slightly dilated.

Hunt and Price-Jones found that whereas in the cardiac group of cases the number of red corpuscles and the percentage of haemoglobin are normal in cases with nocturnal attacks of dyspnoea they are always raised, the average percentage of haemoglobin being 110 in one case the number of red corpuscles present per c.mm. was 6 900 000 and the haemoglobin percentage was 118. The red corpuscles vary in size, many being abnormally small basophilia is present but none is nucleated. It is not known how long the change persists, but no diminution had occurred when a second examination was made fifty days after the first. There is no leucocytosis, but a relative increase of small lymphocytes is present. This was also observed by Miller and Rainy (1917) who found that it persisted for eighteen months or more if respiratory digestive or nervous symptoms were present. The blood changes afford important evidence in support of the genuineness of persisting incapacity after gassing, especially if the percentage of lymphocytes is so high that it approaches that of the polymorphonuclear cells.

The frightful experience of being gassed often leaves its effects upon a man's nervous system, and some of the dyspnoeic attacks are certainly aggravated by apprehension.

Dogs and cats exposed to drift gas became very restless, but appeared to be otherwise unharmed. Horses were much more sensitive and many died.

### Prognosis.

Nothing is known as to the proportion of men who die from "gassing" on the field. Before efficient respirators were in use about 5 per cent of those who reached the clearing stations died within forty-eight hours. Of those who reached the base hospitals between 1 and 2 per cent died in the second or third week from broncho-pneumonia or other pulmonary complications. The mildest cases are often fit for light duty after a short period of rest, but they should not be sent back until all adventitious sounds have disappeared from the lungs. A considerable time elapses before complete restoration of health occurs in the more severe cases, but, contrary to what might have been expected, no permanent ill-effects occurred, even in men who had suffered from extreme cyanosis with acute pulmonary oedema. Many of the latter returned to full military duty after three or four months.

Two-thirds of the patients are fit for discharge from hospital in about two months. Hunt and Price-Jones have shown that some idea of the prognosis can be obtained by a walking test. If the pulse has not fallen below 88 within five minutes after walking half a mile in ten minutes, at least six weeks will be required for recovery. In a paroxysmal case if the hæmoglobin exceeds 104 per cent of the normal, the man will be unfit for at least two months. The difference between the rate of the pulse on lying and sitting up and the presence or absence of functional murmurs are of no importance in prognosis. A man may finally be regarded as fit for light duty if he can march three miles at the normal rate without exhaustion, dyspnoea or vertigo. In a few cases the heart is so greatly dilated that it is obvious that several months must pass before he will be able to return to duty.

A small proportion of patients developed progressive dyspnoea with recurrent bronchitis as a result of diffuse fibrosis and emphysema of the lungs. This was probably a result of the secondary infective broncho-pneumonia and was not directly due to irritation by gas. There is no evidence that gassing predisposed to pulmonary tuberculosis.

### Prophylaxis

The introduction of efficient respirators in 1916 almost abolished the danger of drift gas. Regular drill in the use of the respirators and inspection to see that they are in good condition are most important, as it takes time to get accustomed to breathing whilst wearing a respirator and a damaged respirator may be worse than useless.

### Treatment

The patient should be kept warm with extra blankets, hot-water bottles and hot drinks, and his bed should be near an open window or out of doors. Owing to the acute gastritis often present a fluid diet should be given at first. Absolute rest in the recumbent position is of the greatest importance.

In severe cases an effort should at once be made to expel the fluid, which is drowning the patient, from the lungs, and to allow the air to escape from the over-distended portions of the lungs by forcing it through the obstructed bronchi. This can be done by artificial respiration repeated whenever the dyspnoea becomes extreme.

Unless the patient is collapsed or unconscious, vomiting gives great relief by expelling large quantities of yellowish frothy fluid from the lungs. If this does not occur spontaneously, the patient should put his finger down his throat after drinking half a pint of warm salt water.

The inhalation of oxygen relieves cyanosis and enormously improves the patient's condition. As it is very difficult to get a patient who is fighting for breath to tolerate any form of mask, the easiest way to administer oxygen is by a double nasal tube. Even a single nasal tube proved extremely effective in the last war. The oxygen should be given continuously until its withdrawal no longer leads to a recurrence of cyanosis.

Theoretically atropine should help to diminish bronchial spasm and secretion during the first twenty four hours but it was found useless in severe cases and disappointing in slighter ones.

When the acute symptoms have abated in severe cases and from the onset in slight cases ammonium carbonate should be given in doses of gr. v every three hours. It produces copious expectoration, which results in improvement of colour and considerable relief. When great restlessness and mental distress are present, morphia, gr  $\frac{1}{4}$  should be injected.

Extreme cyanosis with a full pulse is greatly relieved by bleeding

breathing becomes easier, headache is relieved, and the patient falls into a refreshing sleep. The effect is most marked if venesection is performed in the first few hours. From 15 to 25 oz. of blood should be slowly removed. The blood is dark and coagulates with abnormal rapidity. Bleeding is contra-indicated if the patient is pale and collapsed.

After the acute stage has passed the patient should not be kept in bed longer than is absolutely necessary. The cardiac group of after-symptoms is, like the effort syndrome resulting from other causes, greatly benefitted by graduated exercises. In cases with paroxysmal dyspnoea associated with excess of red corpuscles in the blood, much improvement follows periodic venesection (Hunt and Price-Jones).

## VESICANT GASES

### MUSTARD GAS AND LEWISITE

Mustard gas or dichloro-diethylsulphide,  $S \begin{matrix} \diagup CH_2 CH_2 Cl \\ \diagdown CH_2 CH_2 Cl \end{matrix}$ , the

yellow cross gas of the Germans and ypérite of the French, was the most effective gas used in the last war. It accounted for 77 per cent of the 160,970 cases of gas poisoning admitted to the casualty clearing stations of the British Expeditionary Force. In the crude form it is a heavy dark oily fluid with a smell resembling that of mustard or garlic, but it is not chemically related to oil of mustard. It may cause casualties in concentrations insufficient to produce more than a very faint odour, and this may be completely masked by smoke or fumes from high explosive shells. The sense of smell to mustard gas is quickly fatigued so that the odour after a short time is no longer appreciated, but if a respirator is worn the smell is recognised each time it is removed. The boiling point of the oil is high (217° C.) and the vapour pressure low, its low vaporisation at ordinary temperatures and its stability render it extremely persistent and all kinds of articles may remain dangerous to handle for long periods after contamination. As it freezes at 6° C. it cannot be used in cold weather. It is almost insoluble in water but freely soluble in animal oils, and it consequently finds an easy entry into the skin. It has remarkable powers of penetrating materials, such as clothing, leather, wood and bricks. Decontamination is consequently very difficult.

Mustard gas is absorbed by the lipid of the tissues with extreme rapidity and exerts an immediate destructive action on the cells. The changes are progressive, but lead to no symptoms until after a latent period of varying duration. In spite of this latent period the destructive action is immediate. Thus if both eyes of a rabbit are exposed simultaneously to liquid mustard gas and one is immediately irrigated, they are none the less indistinguishable from each other the next day. The irritant action of the gas on the tissues does not itself give rise to any pain. The lesion which slowly develops as a result of the penetration and destructive action of the poison is the sole cause of symptoms. When the concentration of the vapour is low prolonged or repeated exposure may finally cause symptoms. In the last war mustard gas was released as vapour from shells, but in the present war direct splashing with the liquid dispersed by aircraft from containers or as a spray may be expected. The latent period would be much reduced and the effects would be much more severe. Fortunately however improved methods of distributing the poison are to a great extent neutralised by improved protective methods.

Lewisite chlorvinyl-dichloroarsene, has properties similar to those of mustard gas but as it is easier to detect and is less persistent it is unlikely to replace mustard-gas in chemical warfare. It was not ready for use until the end of the last war so that its toxic properties are known only from laboratory experiments.

### Symptoms

Symptoms do not develop at once. After a varying period the eyes, stomach, skin and upper respiratory tract are successively affected, though in mild cases one or more of them may escape. The symptoms are all caused by the direct irritant action of the mustard-gas and there is no general toxæmia. The damaged tissues are very liable to become infected and septic burns and bronchopneumonia may cause severe toxæmia.

*Eyes*—After an interval of two to forty-eight hours, but commonly three to twelve, which varies with the concentration and duration of exposure to the gas, the patient feels a smarting sensation in his eyes quickly followed by profuse lachrymation, photophobia, pain and headache. The eyeballs rapidly swell till the palpebral fissure is closed. Photophobia is extreme, and attempts to separate the lids produce blepharospasm and pain. When fully developed the pain is rarely severe, and apart from the muco-purulent secretion there is very little lachrymation.

The conjunctiva is at first congested and quickly becomes extremely cedematous. The swollen conjunctiva on each side of the cornea forms a yellow-white opaque band projecting between the eyelids, and sometimes two additional folds project above and below from under the eyelids

The cornea is grey and hazy and its surface lustreless. Ulceration may follow and in exceptional cases permanent opacities are produced

In mild cases the symptoms subside in two or three days. As the cedema disappears, the white interpalpebral area becomes congested and the congested areas protected by the eyelids regain their normal colour

Mild cases, which amount to 75 per cent. of the total, are fit for duty in two weeks. In the 15 per cent. intermediate cases, four to six weeks are required for recovery, and in the remaining 10 per cent. severe cases with corneal ulceration as much as four months may be required. Predisposition to recurrent inflammation may remain, especially with exposure to wind, dust and glare in the tropics

In 1940 Phillips drew attention to the occasional delayed action of mustard gas. The patients, of whom he was able to collect no fewer than seventy at Moorfields, gave a history of having been unable to open their eyes for about a week after gassing, the immediate effects of which were so severe that they had remained under treatment in hospital for the unusually long period of four to six months. They then remained free from trouble from six to sixteen years, when lachrymation, photophobia or failing vision developed.

The recurrent ulceration which ensued was most marked in the lower third of the cornea, especially in the 5 and 7 o'clock areas. The condition has recently been investigated by Ida Mann and Pullinger by experiments on rabbits and observations on patients whose eyes had been accidentally injured in laboratories. The original injury to the eye is followed by a very slow regenerative process, in which the cornea is invaded by newly formed blood vessels of extremely abnormal form. In the course of years the vessels regress, cholesterol is deposited and fatty degeneration occurs. These changes are responsible for the delayed ulceration. During the long latent period there are no symptoms, though diagnostic signs can be recognised throughout with the aid of the slit-lamp. The individual ulcers heal in a few days, but the recurrent ulceration leads to increasing deterioration of vision. In 1940 there

were 51 blinded men and 180 others still receiving pensions on account of injury caused by mustard gas to their eyes in the war of 1914-18.

*Skin.*—The areas of skin most affected are the exposed parts—face and hands and the moist parts—the axillæ, genitals and groins. There is generally a latent period of about twelve hours. In slight cases it may be as long as four five or more days. In such cases nothing more than a diffuse erythema is produced, together with œdema where the subcutaneous tissue is loose, as in the eyelids and scrotum. There is at first some irritation but no pain. When the burn is extensive fever and mild general toxic symptoms occur. After forty-eight hours the red colour changes to lilac and then to copper and finally brown. Later the skin desquamates and the pigmentation disappears.

In more severe cases the skin may be affected as early as the eyes. Small vesicles appear on the erythematous skin during the next forty-eight hours. They may coalesce to form bullæ and finally large irregular blisters with inflamed base at first red and later copper coloured. The contents of the blisters almost always become infected and their bases form indolent ulcers, which finally heal to leave permanent scars.

If liquid mustard gas is spattered directly on the skin deep burns are produced.

*Respiratory system.*—Any time from two to four days after exposure the nose, pharynx and larynx, and to a less extent the trachea and large bronchi become inflamed, but the small bronchi always escape. The condition varies from a mild catarrh to extensive necrosis and sloughing with secondary infection. Membranes may form and cause obstruction, and casts of necrotic epithelium from the trachea and bronchi may be coughed up.

The nose may be affected as early as the eyes. The mucous membrane becomes acutely inflamed as a result of irritation by the gas it is then invaded by the normal bacteria of the nose. An acute rhinitis develops, the symptoms of which differ in no way from those of an exceptionally bad cold. The patient sneezes and there is an abundant watery discharge, which quickly becomes mucopurulent. The anterior nares become very sore and encrusted. In exceptionally severe cases ulceration and hæmorrhage may occur.

The pharynx is inflamed and may cause swallowing to be painful and difficult. Laryngitis gives rise to hoarseness and aphonia. In

the early stages there may be painful paroxysms of coughing with no expectoration. In severe cases, where there has been exposure to a high concentration of gas, the vocal cords are superficially ulcerated and may become cedematous. Complete recovery generally occurs within a fortnight; persistence of aphonia after this is almost always due to hysteria.

Two or three days after the onset of laryngitis, if tracheitis and bronchitis develop, the dry irritating cough is replaced by a loose cough with abundant muco-purulent expectoration, pain is felt behind the sternum, and the temperature rises. The bronchitis generally clears up completely in four to six weeks and leaves no after-effects.

Occasionally secondary infection gives rise to septic bronchitis and broncho-pneumonia. In rare cases pulmonary abscess, bronchiectasis or gangrene of the lung develops.

*Stomach*—From the fourth to the eighth hour epigastric pain, nausea and vomiting may occur owing to irritation of the stomach by the swallowing of mustard gas dissolved in saliva and nasal secretion. The bowels are generally unaffected, but in severe cases there may be diarrhoea with excess of blood-stained mucus in the stools resulting from enteritis. These symptoms rarely persist for more than forty-eight hours.

### **Treatment.**

Except in severe cases the patient should not be kept in bed for more than two or three days. He should be sent out of doors and encouraged by exercise and discipline to realise that no serious harm has been done and that gassing does not give rise to any lasting ill-effects.

*Eyes*—"No first-aid treatment can compete with Nature's rapid and complete removal of unabsorbed mustard gas—reflex action of the lids and immediate flow of tears," and no treatment will influence the development of a lesion in the damaged cells (Poole, 1937). Treatment must be palliative,—to prevent infection of the devitalized tissue, allay irritation and control spasm. As soon as it is known that exposure to mustard gas has occurred, the eyes should therefore be washed with warm normal saline solution every two hours, even if there are as yet no symptoms. As free drainage is important the eyes should be shaded, but not bandaged. A few drops of sterilized liquid paraffin should be inserted between the lids several times daily to prevent them from adhering together. In

most cases no further treatment is required. In severe cases in which the discharge becomes muco-purulent 2 per cent. argyrol solution should be applied twice daily and if pain and blepharospasm are severe 1 per cent. atropine solution should be used twice a day but cocaine should be avoided because of its devitalizing action on the corneal epithelium. The patient should not be kept in the dark or wear dark glasses or a shade when the conjunctivitis is no longer acute, or hysterical symptoms are likely to develop. From the onset he should be told that there is no reason to worry as recovery will be rapid and complete.

In cases of delayed ulceration of the cornea great improvement in visual acuity follows the use of contact lenses owing to the substitution of the smooth glass for the uneven surface of the cornea as the main refractive surface. The lenses should be fitted as early as possible, as they afford a certain means of protecting the insensitive and ill nourished cornea and preventing recurrent ulceration (Phillips).

*Skin.*—In the early stages of erythema the skin should be washed with soap and water and the pubic and axillary hair cut short. A lotion containing 1 per cent. tannic acid and 4 per cent. calamine and zinc oxide may be applied to allay irritation. Blisters should be emptied by means of a hypodermic syringe. The intact epithelium is then allowed to collapse on to the raw surface beneath. The evacuation of the blisters may require to be repeated by the application of gentle pressure after puncturing, as serum continues to ooze. With small burns the dead skin can be removed and cod liver oil is then applied on lint. The dressings are changed daily. With larger burns the confluent blisters are emptied and the loose epithelium removed. Three layers of lint soaked in a mixture of one part of dettol with five parts of 5 per cent. tannic acid solution are then applied and covered lightly with wool. The bandage and wool are removed every two hours and the lint is again soaked with the solution. At the end of twelve hours the lint is removed. A firm coagulum has formed by this time it is sprayed with 5 per cent. tannic acid and dried. The surrounding skin is swabbed with the same solution. If the skin is severely infected continuous mild antiseptic baths are effective. Hot hip baths of normal saline solution allay the intense irritation when the groin and genitals are affected.

*Respiratory tract.*—Pain and discharge from the nose requires

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# INDEX

- Abdominal distension following dysentery 195
- Alcohol and effort syndrome, 204
- war neuroses, 3
- Amnesia, following concussion, 122, 123
- Amoebic dysentery 301
- hepatic abscess, 311
- hepatitis, 311
- Anaphylaxis, 298, 425
- Anxiety neuroses, 149
- Aphasia, diagnosis from hysterical mutism, 70
- Aphonia, hysterical, 72
- Appendicitis and amoebic dysentery 308
- malaria, 354
- trench fever 224
- Arthritis, dysenteric, 287
- Asphyxiant gases, 493
- Asthenopia, functional, 112
- Bacillary dysentery 284
- Bent back of soldiers, 51
- Blackwater fever 368
- Blast, 121
- Blepharospasm, hysterical, 102
- Blindness, hysterical, 99
- Blood pressure in soldiers, 145
- Camptocormia, 51
- Catarrhal jaundice, 336
- Cerebro-spinal meningitis, 380
- Chlorine poisoning, 493
- Colitis, post-dysenteric, 320
- Colon, irritable, following dysentery 320
- Concussion, cerebral, 121
- spinal, 125
- Constipation in soldiers, 196
- Contractures, hysterical, 21
- reflex, 21
- Convulsions, hysterical, 65
- Cordite and effort syndrome, 204
- Deafness, hysterical, 84
- Dengue fever 230
- Dermatophytosis, 472
- Desert sore, 400
- Dhobie's itch, 472
- Diabetes, 148
- Diaphragm hysterical spasm of, 195
- Diarrhoea, post-dysenteric, 320
- Diphtheria, 392
- Diseases, skin, 450
- Distension, abdominal, following dysentery 195
- Dumbness, hysterical, 69
- Duodenal ulcer 183
- Dysentery amoebic, 301
- bacillary 284
- malarial, 352
- Dyspepsia, functional, 190
- Effort syndrome, 199
- Enteric fever 261
- Epidemic jaundice, 323
- Epilepsy 67
- Exhaustion, 136
- Fever blackwater 368
- meningococcal, 376
- paratyphoid, 281
- trench, 216
- typhoid, 261
- typhus, 235
- Fits, epileptic, 67
- hysterical, 65
- uræmic, 441
- Flies and dysentery 303
- Functional dyspepsia, 190
- nervous disorders, 1
- Galta, hysterical, 45
- Gas; mustard, 500
- Gas-poisoning, 492
- functional sequelæ of, 7
- tear, 506
- Gastric ulcer 183
- Gastritis, 180
- Giardia intestinalis*, 318
- Hæmatomyelia, hysterical paralysis following, 132
- Hair blanching of, 147
- erection of, 146

*Effort syndrome.*—After exposure to chlorine and phosgene and less frequently to mustard gas, symptoms of effort syndrome sometimes developed, especially in patients who were evacuated to England.

*Anxiety symptoms*—The terror produced by exposure to gas in war-worn soldiers may be the final factor leading to a breakdown with the development of anxiety symptoms

Recognition of the possibility of the development of the various neurotic symptoms following gassing by medical officers in the hospitals in which they are first received should prevent their development

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## INDEX

- Abdominal distension following dysentery 195
- Alcohol and effort syndrome, 204
- war neuroses, 3
- Amnesia, following concussion, 122, 123
- Amoebic dysentery 301
- hepatic abscess, 311
- hepatitis, 311
- Anaphylaxis, 298, 425
- Anxiety neuroses, 149
- Aphasia, diagnosis from hysterical mutism, 70
- Aphonia, hysterical, 73
- Appendicitis and amoebic dysentery 308
- malaria, 354
- trench fever 224
- Arthritis, dysenteric, 287
- Asphyxiant gases, 483
- Asthenopia, functional, 112
- Bacillary dysentery 284
- Bent back of soldiers, 51
- Blackwater fever 368
- Blast, 121
- Blepharospasm, hysterical, 102
- Blindness, hysterical, 99
- Blood pressure in soldiers, 145
- Camptocormia, 51
- Catarrhal jaundice, 336
- Cerebro-spinal meningitis, 380
- Chlorine poisoning, 493
- Colitis, post-dysenteric, 320
- Colon, irritable, following dysentery 320
- Concussion, cerebral, 121
- spinal, 125
- Constipation in soldiers, 196
- Contractures, hysterical, 21
- reflex, 21
- Convulsions, hysterical, 65
- Cordite and effort syndrome, 204
- Deafness, hysterical, 84
- Dengue fever 230
- Dermatophytosis, 472
- Desert sore, 400
- Dhobie's itch, 472
- Diabetes, 148
- Diaphragm hysterical spasm of 185
- Diarrhoea, post-dysenteric, 320
- Diphtheria, 392
- Diseases, skin, 450
- Distension, abdominal, following dysentery 195
- Dumbness, hysterical, 69
- Duodenal ulcer 183
- Dysentery amoebic, 301
- bacillary 284
- malarial, 353
- Dyspepsia, functional, 190
- Effort syndrome, 199
- Enteric fever, 261
- Epidemic jaundice, 323
- Epilepsy 67
- Exhaustion, 136
- Fever blackwater 368
- meningococcal, 378
- paratyphoid, 261
- trench, 216
- typhoid, 261
- typhus, 235
- Fits, epileptic, 67
- hysterical, 65
- uramic, 441
- Flies and dysentery 303
- Functional dyspepsia, 190
- nervous disorders, 1
- Gaita, hysterical, 45
- Gas; mustard, 500
- Gas-poisoning, 482
- functional sequelae of, 7
- tear, 506
- Gastric ulcer 183
- Gastritis, 180
- Giardia intestinalis*, 318
- Hæmatomyelia, hysterical paralysis following, 122
- Hair, blanching of, 147
- erection of, 146

- Head injuries, 121
- Headache, 124
  - neurasthenic, 139
- Hearing disorders of, 84
- Heart, soldiers', 199
- Hepatic jaundice, infective, 323
- Hiccup, hysterical, 81
- Hyperacousis, 96
- Hyperadrenalism, 143
- Hyperthyroidism, 143
- Hypnotism, 12
- Hysteria, 5
- Hysterical aphonia, 72
  - blindness, 99
  - contractures, 21
  - deafness, 84
  - dumbness, 69
  - fits, 65
  - gaits, 45
  - hiccup, 81
  - idioglossia, 82
  - mutism, 69
  - paralysis, 14
  - postures, 45
  - stammering, 77
  - tremor, 61
  - vomiting, 193
- Hystero epilepsy, 65
- Idioglossia, hysterical, 82
- Impetigo contagiosa, 485
- Incontinence of urine, hysterical, 134
- Infective hepatitis 323
- Influenza diagnosis from typhoid
  - and paratyphoid fever, 275
- from trench fever, 230
- Insomnia, 157
- Intervertebral disc, prolapse of, and sciatica, 45
- Irritable colon following dysentery, 320
- Irritants, nasal, 507
- Jaundice, catarrhal, 336
  - epidemic, 323
  - leptospiral, 329
- Kala azar, diagnosis from malaria, 335
- Lachrymator gases, 506
- Lambia diarrhoea, 318
- Leptospiral hepatitis, 329
- Lewisite, 500
- Lice, 464
  - and skin, 463
  - and trench fever, 216
  - and typhus, 235
- Localized tetanus, 430
- Lumbago, 45
- Malaria, 343
- Malingering, diagnosis of, 19
- Melanoderma following pediculosis, 467
- Meningococcal fever, 376
  - meningitis, 380
  - septicæmia, acute, 379
  - — chronic, 384
- Migraine, 112
- Mustard gas, 500
- Mutism, hysterical, 69
- Nasal irritants, 507
- Nephritis, in leptospiral jaundice, 333
- war, 437
- Nervous disorders, functional, 1
- Neurasthenia, 136
- Neuroses, anxiety, 149
- Nightmares, war, 157
- Paralysis, hysterical, 13
  - reflex, 22
- Paraplegia, hysterical, 13, 130
- Paratyphoid fever, 261
- Pediculosis corporis, 463
- Phosgene poisoning, 493
- Poisoning, chlorine, 493
  - gas, 492
  - mustard gas, 500
- Postures, hysterical, 45
- Pseudo-flatulence, 195
- Ptosis, hysterical, 101
- Reflex contractures, 22
- Relapsing fever, 230
- Retention of urine, hysterical, 134
- Ringworm, 472
- Scabies, 451
- Sciatica, 45
- Shell-shock, 121
- Skin diseases, 450
- Soldier's heart, 199
- Speech, disorders of, 68

Spine, concussion of, 125  
Spirochaetal jaundice, 329  
Stammering, hysterical, 77  
Stupor following concussion, 122  
— functional, 113

Tear gases, 506  
Tetanus, 413  
— localised, 430  
Tinea cruris, 472  
Tinnitus, 98  
Tremor in hyperthyroidism 147  
— hysterical, 61  
— neurasthenic, 139  
Trench fever 216  
— foot, 415  
— nephritis, 437

Trench shin, 223  
Tropical ulcer 407  
Typhoid fever 261  
Typhus fever 235

Ulcer gastric and duodenal, 183

*Varicella gangrenosa*, 399  
Veldt sore, 400  
Vision, hysterical disorders of, 93  
Vomiting, hysterical, 193

War nephritis, 437  
— neuroses, 1  
Weigl's disease, 217  
Wounds and hysteria, 8